A Dissertation on

EFFICACY OF TRANSVERSE CEREBELLAR DIAMETER / ABDOMINAL CIRCUMFERENCE RATIO - A GESTATIONAL AGE INDEPENDENT PARAMETER IN ASSESSING FETAL GROWTH RESTRICTION

Dissertation submitted to

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BONAFIDE CERTIFICATE

Certified that this dissertation is the bonafide work of Dr.C.SARITHA on **"EFFICACY OF TRANSVERSE CEREBELLAR DIAMETER / ABDOMINAL CIRCUMFERENCE RATIO - A GESTATIONAL AGE INDEPENDENT PARAMETER IN ASSESSING FETAL GROWTH RESTRICTION"** during her M.D., (Obstetrics & Gynaecology) course from April 2010 to April 2012 at the Stanley Medical College and Raja Sir Ramasamy Mudaliar Lying-in Hospital, Chennai.

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DECLARATION

I solemnly declare that the dissertation titled **"EFFICACY OF TRANSVERSE CEREBELLAR DIAMETER/ABDOMINAL CIRCUMFERENCE RATIO - A GESTATIONAL AGE INDEPENDENT PARAMETER IN ASSESSING FETAL GROWTH RESTRICTION"** is done by me at RSRM Lying in Hospital, Stanley Medical College and Hospital, Chennai during November 2010 to October 2011 under the guidance and supervision of **Prof. V. Kalaivani, M.D., D.G.O.,** Professor and Chief of the Department of Obstetrics and Gynaecology, Stanley Medical College & RSRM Lying in Hospital, Chennai-13.

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CONTENTS

ABBREVIATIONS

AC	**-**	**Abdominal Circumference**
APGAR	**-**	**Appearance, Pulse, Grimace, Activity, Respiration**
AGA	**-**	**Appropriate for Gestational Age**
BPD	**-**	**Biparietal Diameter**
CHT	**-**	**Chronic Hypertension**
FGR	**-**	**Fetal Growth Restriction**
FL	**-**	**Femoral Length**
GA	**-**	**Gestational Age**
GDM	**-**	**Gestational Diabetes Mellitus**
HC	**-**	**Head Circumference**
LGA	**-**	**Large for Gestational Age**
LSCS	**-**	**Lower segment caesarean section**
NICU	**-**	**Neonatal Intensive Care Unit**
SGA	**-**	**Small for Gestational Age**
TCD	**-**	**Transverse Cerebellar Diameter**
GCK	**-**	**Glucokinase**
HNF1 beta	**-**	**Hepatocyte nuclear factor *1beta***
HNF4 alpha	**-**	**Hepatocyte nuclear factor 4 alpha**
ADCY5	**-**	**Adenylate cyclase type 5**
CCNL1	**-**	**Cyclin-L1**
CPM	**-**	**Confined Placental Mosaicism**
CRH	**-**	**Corticotrophin Releasing Hormone**
SD	**-**	**Standard Deviation**
IUGR	**-**	**Intrauterine Growth Restriction**
PPV	**-**	**Positive Predictive Value**
NPV	**-**	**Negative Predictive Value**

Introduction

INTRODUCTION

The Process of birth is the most dangerous journey an individual undertakes. A healthy new born is the goal of every expectant mother and her obstetrician.

It is estimated that the incidence of fetal growth restriction is 3-10% [1]. Fetal growth restriction is associated with substantial perinatal morbidity and mortality. Fetal demise, birth asphyxia, meconium aspiration, neonatal hypoglycemia and hypothermia are all increased in growth restriction. In addition it has been also found that these growth restricted infants have increased 1year infant mortality rate [2] and abnormal neurological development. The desire to prevent such mal occurrences during pregnancy has prompted the clinician to develop various methods of assessing the fetal condition in utero. Ideally the best investigation must be simple, safe, reproducible, reliable, non- invasive and accurate and should cause no damage to the mother and fetus. Prenatal ultrasonography, which fulfills almost all these prerequisites plays an important role in antepartum fetal surveillance. An accurate determination of gestational age, identification of major congenital anomalies, evaluation of fetal growth and assessment of fetal well being and maturity are all possible due to the availability of ultrasound.

The assessment of fetal growth is important to the provision of optimum prenatal care. As the clinical estimation of the fetal growth is not reliable, prenatal ultrasonography provides an opportunity to more accurately assess the fetal growth. The most commonly used parameters to evaluate fetal growth are biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC) and femur length (FL). Of all the ultrasound derived biometric parameters, the AC seems to be the best predictor of fetal growth restriction (FGR). But all these parameters can be correlated only if gestational age is accurately known. But uncertainty of the gestational age occurs frequently and makes the differentiation between the appropriate for gestational age and the small for gestational age fetus difficult.

Measurement of transverse cerebellar diameter (TCD) is an accurate method of estimating gestational age in cases of uncertain dates and even in dolicocephaly or brachycephaly where biparietal diameter could not be used. It has been proposed that TCD is not affected in fetal growth restriction because of the brain sparing effect[3]. Fetal AC is affected early in the process of growth restriction[4]. Hence, TCD/AC ratio increases in fetal growth restriction which fairly remains constant throughout normal pregnancy[5]. TCD/AC ratio may convey more precise

information regarding the fetal growth and development than bony measurements of the fetal head and AC alone.

This study was primarily planned to study the TCD among pregnant women and to find whether TCD/AC ratio can diagnose fetal growth restriction.

Aims and Objectives

AIMS AND OBJECTIVES OF THE STUDY

1. To evaluate the validity of TCD/AC ratio in diagnosing fetal growth restriction.
2. To find out the cut-off value of TCD/AC ratio for diagnosis of fetal growth restriction.

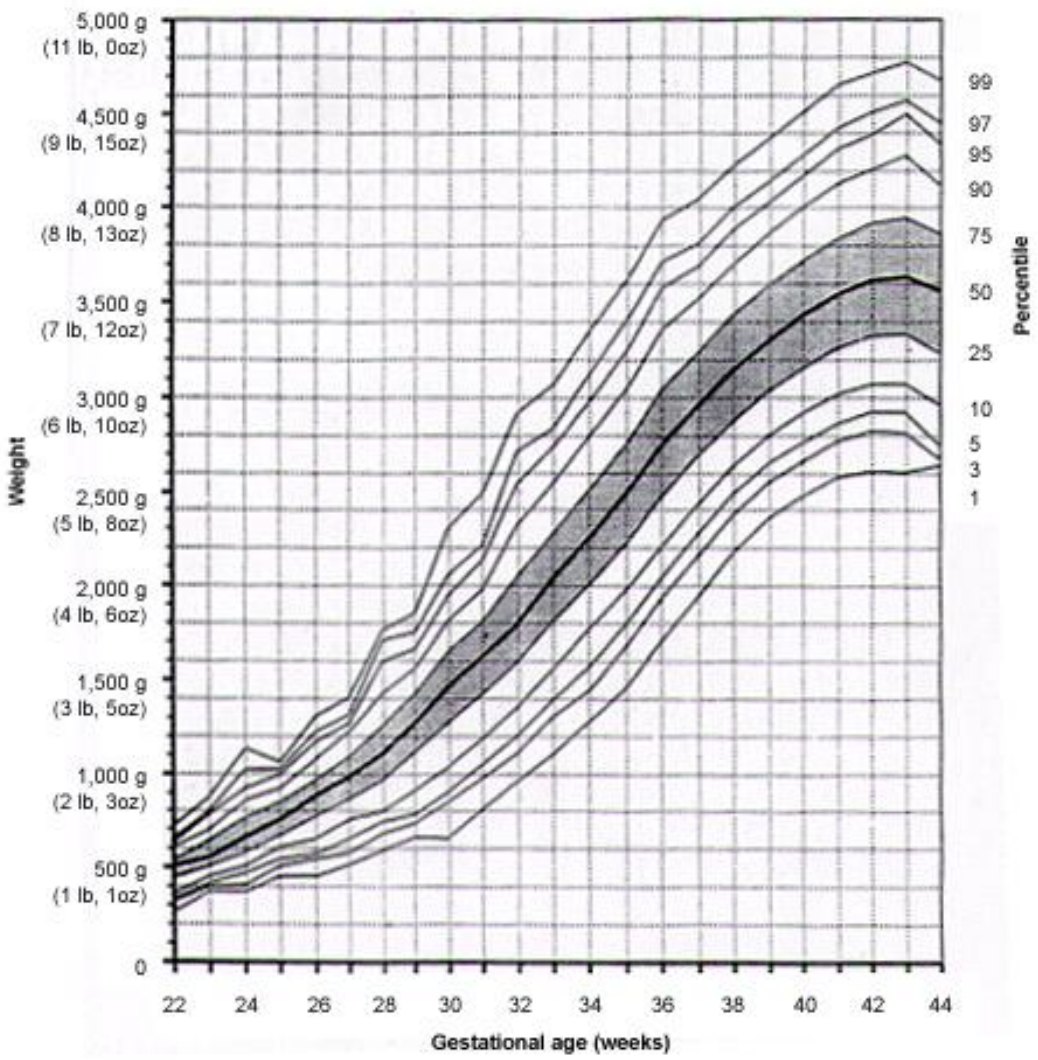
Review of Literature

REVIEW OF LITERATURE

Fetal growth restriction can be defined as a condition where the fetus fails to achieve its genetic growth potential and consequently is at risk of increased prenatal morbidity and mortality[6]. Incidence of FGR is approximately 5% in general population. However, the incidence varies depending on the population under examination and the standard growth curves used as reference.

Birth weight is usually taken as the sole criterion to assess fetal growth and consequently fetuses with a birth weight less than the 10th percentile of those born at the same gestational age, or two standard deviations below the population mean are considered growth restricted. However, this definition does not make a distinction among infants who are constitutionally small, growth-restricted and small, and not small but growth-restricted relative to their potential. As an example, as many as 70 percent of fetuses who weigh below the 10th percentile for gestational age are small simply because of constitutional factors such as female sex or maternal ethnicity, parity, or body mass index; they are not at high risk of perinatal mortality or morbidity [7]. Therefore the term FGR refers to fetuses that are small for gestational age with features of chronic hypoxia or failure to thrive. Moderate and severe FGR are defined as birth weight in the 3rd to 10th percentile and less than 3rd percentile, respectively.

Growth percentiles for fetal weight versus gestational age



Normal term infants typically weigh more than 2500 g by 37 weeks gestation.

1. NORMAL GROWTH

The process of fetal growth comprises three consecutive and somewhat overlapping phases. The first phase is the phase of cellular hyperplasia and encompasses the first 16 weeks of gestation. The second phase, known as the phase of concomitant hyperplasia and hypertrophy, occurs between the 16^{th} and 32^{nd} weeks and involves increases in cell size and number. The third phase, called the phase of hypertrophy, occurs between 32weeks and term and is characterized by a rapid increase in cell size.

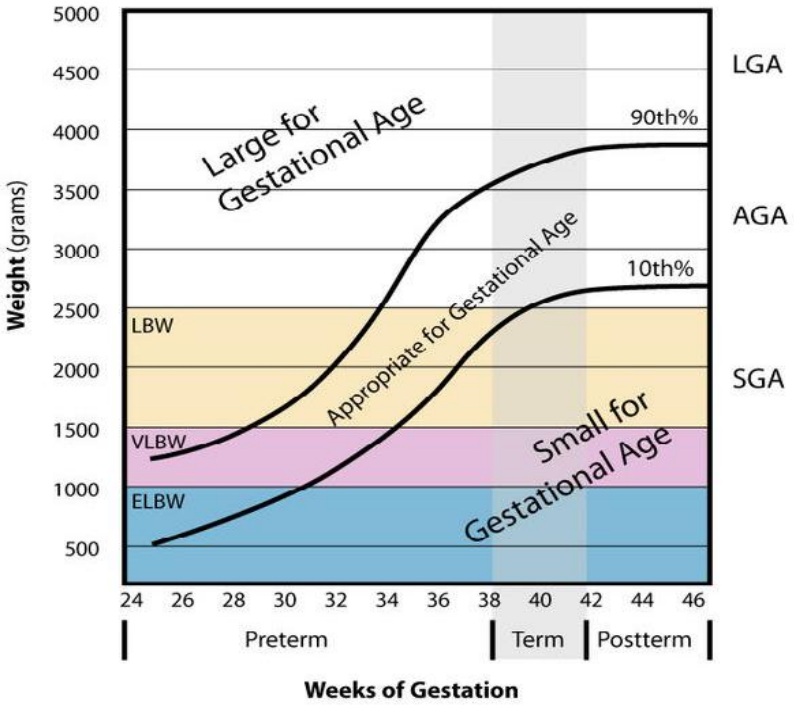
2. CLASSIFICATION OF FETAL GROWTH RESTRICTION

Campbell and Thoms (1997) described the use of head-to-abdomen circumference ratio (HC/AC) to differentiate growth restricted fetuses. Those who were symmetrical were proportionally small, and those who were asymmetrical had disproportionally lagging abdominal growth.

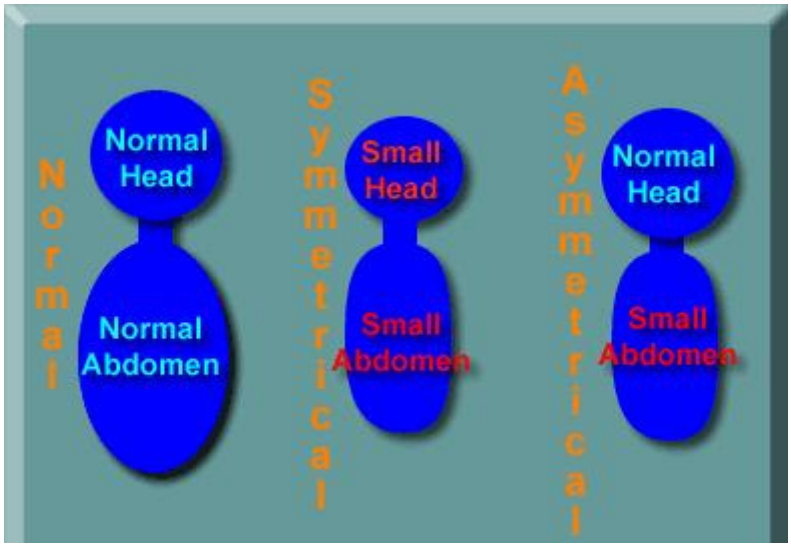
SYMMETRICAL FGR

Symmetrical FGR occurs as a result of early fetal insult in the pregnancy. This early fetal insult results in relative decrease in cell number and size. Symmetrical FGR accounts for 20-30% growth

In utero Growth Status according to Birthweight percentile



Fetal Growth Restriction



restricted fetuses. The causes of symmetrical FGR include viral infections, chemical exposure or cellular maldevelopment with aneuploidy may cause a proportionate reduction of both head and body size.

All parameters, i.e. head and abdominal circumference, length and weight, are below the 10^{th} percentile for gestational age, hence these infants have a normal ponderal index.

Medical interventions to improve the fetal growth are rarely effective as the causative factor is usually uncorrectable[8].

ASYMMETRICAL FGR

Asymmetrical FGR follows a late pregnancy insult such as uteroplacental insufficiency. It is usually associated with maternal diseases such as chronic hypertension, renal disease, and vasculopathy. Resultant diminished glucose transfer and hepatic storage would primarily affect cell size and not number, and fetal abdominal circumference-which reflects liver size-would be reduced. Such somatic growth restriction is proposed to result from preferential shunting of oxygen and nutrients to the brain, which allow normal brain and head growth –so-called brain sparing. The fetal brain is normally relatively large and the liver relatively small. Accordingly, the ratio of brain weight to liver weight during the last 12 weeks, usually about 3to 1, may be

increased to 5 to 1 or more in severely growth restricted fetuses. It accounts for approximately 70-80% of growth restricted fetuses. The Ponderal index is low with low birth weight and abdominal circumference, but normal head circumference and fetal length.

However, in case of severe placental insufficiency the head growth curve may also be flattened eventually and the size may drop below the normal growth curve. This type of growth restriction leads to decreased amniotic fluid, chronic hypoxia and may result in fetal death.

3. ETIOLOGY AND RISK FACTORS :

FGR may be caused by maternal, placental, or fetal factors. Approximately one-third of FGRs are due to genetic causes, and two-thirds are related to the fetal environment. However, no underlying etiology can be identified in at least 40 percent of SGA infants.

FETAL FACTORS

Genetic factors — Population-based intergenerational studies of birth weight have found that genetic factors contribute 30 to 50 percent of the variation in birth weight [9]. Maternal genes influence birth weight more than paternal genes, but both have an effect. Specific allelic variants associated with birth weight include mutations in GCK and HNF1beta, which have been associated with low birth weight, and mutations in

HNF4 alpha, which have been associated with high birth weight. Variants in ADCY5 and loci near CCNL1 also appear to lower birth weight [10].

The susceptibility to FGR is also heritable; in epidemiologic studies, women who were SGA themselves at birth have a two-fold increase in risk of FGR in their offspring [11,12] .Women who give birth to a growth restricted fetus are at high risk of recurrence, and the risk increases with increasing numbers of FGR deliveries.

Chromosome Abnormalities - Karyotypic abnormalities account for up to 20 percent of all FGR [13,14]. The presence of a chromosomal abnormality often results in restriction of fetal growth early in pregnancy; as many as one-quarter of fetuses with early onset FGR have chromosomal abnormalities. Most cases are symmetric, but asymmetric early FGR also occurs [15]. Chromosomal abnormalities associated with FGR include [16]:

- Aneuploidy (e.g. trisomy 18 or 13, Turner 45 X, triploidy)
- Partial deletions (e.g. Cri du chat syndrome 5q, Wolf-Hirschhorn syndrome 4q)
- Ring chromosomes
- Uniparental disomy (e.g. for chromosomes 6, 14, and 16)
- Confined placental mosaicism

- Gene mutations (e.g. mutations in the gene for insulin-like growth factor)

Multiple gestation — Fetal growth in multiple gestations has a direct relationship to the number of fetuses present; the type of placentation also plays a role (monochorionic versus dichorionic). Growth is similar to that of singletons until the third trimester and then slows. The lower weight of fetuses from multiple gestations is thought to be due to an inability of the environment to meet the nutritional needs of multiple fetuses, as well as pregnancy complications more common in multiple gestation (eg, maternal undernutrition, preeclampsia, twin-twin transfusion, congenital anomalies). Placental and umbilical cord anomalies potentially associated with underperfusion (e.g. velamentous cord insertion) are also more common in multiple gestations.

Infection — Infections that develop early in pregnancy have the greatest effect on subsequent growth, but account for less than 5 percent of all cases of FGR. Viruses and parasites (e.g. rubella, toxoplasmosis, cytomegalovirus, varicella-zoster, malaria, syphilis, herpes) may gain access to the fetus transplacentally or across the intact fetal membranes and impair fetal growth by a variety of mechanisms (e.g. cell death, vascular insufficiency). Although uncommon, CMV (Cytomegalo Virus) is the most frequent viral etiology of FGR in developed countries [17].

There is less evidence implicating bacterial infection as an etiology for FGR, although maternal infection with listeria, tuberculosis, chlamydia, and mycoplasma has been reported to increase the risk to FGR.

PLACENTAL FACTORS

Many cases of FGR, particularly recurrent cases, are the result of ischemic placental disease. This term refers to a disease process of the placenta that clinically manifests as preeclampsia, FGR, abruption, or a combination of these disorders[18,19]. All of these disorders may be associated with preterm birth or fetal loss and represent late manifestations of abnormal placental development dating from the earliest stages of pregnancy.

Gross and histological lesions — Any mismatch between fetal nutritional or respiratory demands and placental supply can result in impaired fetal growth. Studies have suggested that there is significant excess placental functional capacity. In sheep models, fetal growth is affected when one-half of the placenta is removed . The human fetus may be more sensitive to a reduction in placental mass: placental weight is 24 percent smaller in growth restricted fetuses than in normally grown fetuses when adjustments are made for gestational age [20].

However, placental functional capacity cannot be accurately assessed from placental weight or dimensions alone. Abnormal

development, narrowing or obstruction of placental vessels, and physical separation at the maternal interface all impair placental function. The types, distributions, and sizes of parenchymal and vascular lesions also play a role; moreover, some maternal disorders (eg, severe maternal malnutrition or alcohol abuse) can affect fetal nutrition without causing a recognizable histopathological lesion [21].

Identifiable placental histological abnormalities associated with fetal undernutrition include abnormalities of the uteroplacental vasculature (maldevelopment, obstruction, disruption), chronic abruption, chronic infectious and idiopathic inflammatory lesions (eg, infection related villitis, chronic villitis of unknown etiology), infarction, distal villous hypoplasia, massive perivillous fibrin deposition (i.e. maternal floor infarction), and thrombosis in the uteroplacental, intervillous and/or fetoplacental vasculature [22].Diffuse chronic villitis of unknown etiology appears to be the most common placental finding in otherwise idiopathic FGR [17,22,23].

Gross placental structural anomalies possibly associated with FGR include single umbilical artery, velamentous umbilical cord insertion, bilobate placenta, circumvallate placenta, placental hemangioma, and, possibly, placenta previa.

Confined placental mosaicism — Confined placental mosaicism (CPM) refers to chromosomal mosaicism (usually involving a trisomy) found in the placenta, but not in the fetus. It occurs significantly more often in the placentas associated with FGR than in controls of normal weight. Approximately 10 percent of placentas associated with idiopathic FGR have been reported to have CPM [24,25]; the rate of CPM in controls undergoing CVS is about 1 percent. The extent of FGR depends upon the chromosomes involved, the proportion of mosaic cells, and the presence of uniparental disomy [26].

Placentas with CPM have a high ratio of placental infarcts and decidual vasculopathy, and one-third of placentas with these findings and FGR have CPM .

MATERNAL FACTORS

Reduction in uteroplacental blood flow — Uteroplacental blood flow may be diminished by faulty development, acquired obstruction, or disruption of the uteroplacental vasculature. Maternal medical disorders (e.g. hypertension, renal insufficiency, diabetes, collagen vascular disease, systemic lupus erythematosus, antiphospholipid syndrome) and obstetrical complications (e.g. preeclampsia) associated with vasculopathy and/or reduced maternal blood volume or blood pressure diminish uteroplacental perfusion and result in FGR [27]. Preeclampsia, in

particular, is characterized by primary failure of trophoblast invasion of the spiral arteries leading to failure of dilatation of these vessels, acute atherosis, occlusion, and infarction.

Constitutionally small mothers

If a women begins pregnancy weighing less than 100 pounds, the risk of delivering an SGA infant is increased at least twofold (simpson and colleagues, 1975). Moreover, intergenerational effects on birthweight are transmitted through the maternal line such that reduced intrauterine growth of the mother is the risk factor for reduced intrauterine growth of her offspring.

Diminished caloric intake — Maternal weight at birth, prepregnancy weight, and weight gain during pregnancy are generally responsible for about 10 percent of the variance in fetal weight [28]. However, severe maternal starvation during pregnancy can have a major impact on fetal growth. As an example, the Dutch population suffered severe famine during the winter of 1944 to 1945; mean maternal caloric intake fell to 450 to 750 kcal a day. As one result of this deprivation, average infant birth weight during this period decreased by 250 grams. Similarly, in Leningrad during the World War II German siege, which resulted in a longer and more profound starvation period (down to 300 kcal of mostly

carbohydrates and no protein), average birth weight fell by more than 500 grams.

Modest degrees of nutritional deficiency also have an effect on birth weight. Women who are underweight at the start of pregnancy or have poor weight gain during pregnancy are at higher risk of delivering an infant weighing less than 2500 grams.

Hypoxemia — Chronic maternal hypoxemia due to pulmonary disease, cyanotic heart disease, or severe anemia is associated with diminished fetal growth. As an example, a study of 96 pregnancies in women with cyanotic congenital heart disease reported that the mean birth weight of full term infants was only 2575 grams, which is significantly lower than the mean birth weight of 3500 grams in the general population [29].

Residing at high altitude also results in a chronic hypoxemic state and lower birth weight. A direct relationship between increasing altitude and lower birth weight has been demonstrated. Birth weight data from 15 areas in Peru located anywhere from sea level to 4575 meters showed birth weight declines an average of 65 grams for every additional 500 meters in altitude above 2000 meters[30]. The fetus can compensate for hypoxemia in a number of ways, including redistribution of circulation to vital organs and deferment of growth, decreased gross body movements, and increasing tissue oxygen extraction. The exact level and duration of

fetal hypoxemia that exceed these compensatory mechanisms are not defined in humans.

Hematological and immunologic disorders — Hematological disorders, such as sickle cell disease, may cause thrombosis of the intervillous space. Autoimmune and alloimmune disorders (e.g. antiphospholipid syndrome) may cause chronic villitis, as well as vasculopathy. Fetal undernutrition and hypoxia are possible sequelae.

Substance use and cigarette smoking — Maternal substance use, including cigarette smoking, alcohol consumption, and illicit drug use can cause FGR either by a direct cytotoxic effect or indirectly from related variables, such as inadequate nutrition.

Smoking during the third trimester appears to have the greatest impact on birth weight; women who quit smoking by the third trimester have birth weights similar to those of nonsmokers [31].

Toxins — Toxic exposures, including various medications such as warfarin, anticonvulsants, antineoplastic agents, and folic acid antagonists, can produce FGR with specific dysmorphic features [32,33].

Fetal exposure to therapeutic, but not diagnostic, doses of radiation can cause permanent restriction of growth. Prepregnancy radiation therapy to the pelvis can result in anatomic changes in the pelvic

vasculature that may lead to reduced fetoplacental perfusion and growth restriction.

Assisted reproductive technologies :

Singleton pregnancies conceived via assisted reproductive technologies have a higher prevalence of both low birth weight and SGA infants compared with naturally conceived pregnancies.

Others:

- FGR is more common among pregnancies at the extremes of reproductive life.
- Uterine malformations may affect uteroplacental perfusion and result in FGR
- A short interpregnancy interval has been associated with low birth weight and FGR, and this may be mediated through a relative depletion in folic acid.
- Chronic maternal stress may also be a factor . Chronic stress is associated with elevated corticotropin-releasing hormone (CRH) levels, which, in turn, may be associated with impaired fetal growth and preterm birth.

4. PATHOPHYSIOLOGY[34]

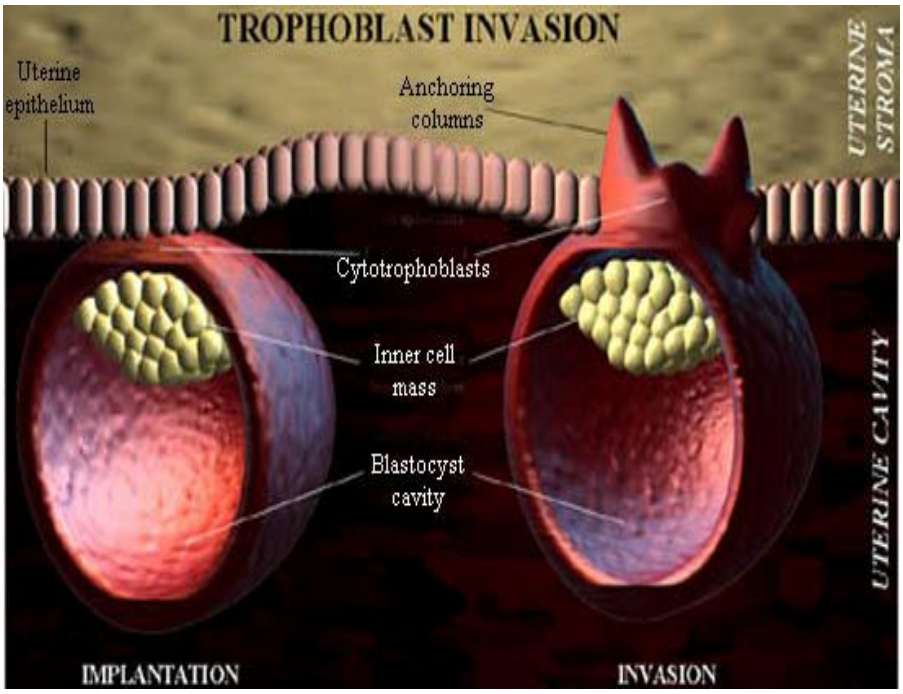
Interference with placental nutrient supply can affect all aspects of placental function. The gestational age at onset, the magnitude of the injury, and the success of adaptive mechanisms determine the ultimate severity. Mild placental disease is more likely to affect organ function and maturation at the cellular level, with little perceivable growth delay perinatally, but may affect adult health (fetal programming), often through epigenetic modifications. With more severe placental disease, fetal growth delay and adaptive organ responses become evident in utero.

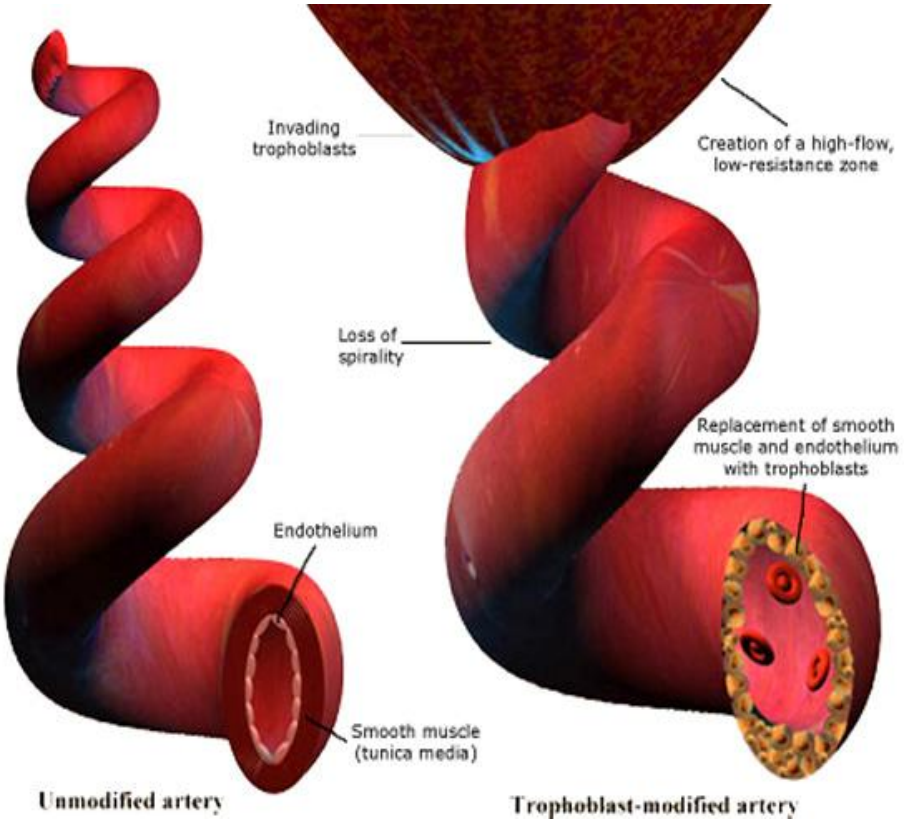
MECHANISMS OF PLACENTAL DYSFUNCTION

The efficiency of maternal to fetal exchange of nutrients, fluid, and waste can become suboptimal when there is a decrease in substrate transporters, an increase in the diffusion distance between maternal and the fetal compartments, a decrease in the exchange area or impedance to blood flow in the maternal and fetal compartments in the placenta. Typically, trophoblast invasion is confined to the decidual portion of myometrium, and the spiral and radial arteries do not transform into low-resistance vessels.

Altered expression of vasoactive substances increases vascular reactivity, and if hypoxia-stimulated angiogenesis is inadequate, placental autoregulation becomes deficient. Maternal placental floor infarcts and

Pathophysiology of FGR





fetal villous obliteration and fibrosis increase placental blood flow resistance, producing a maternal-fetal placental perfusion mismatch that decreases the effective exchange area.

The severity of placental vascular dysfunction is clinically assessed in the maternal and fetal compartments of placenta with Doppler ultrasound. An early diastolic notch in the uterine arteries at 12-14 weeks suggests delayed trophoblast invasion, whereas persistence of “notching” beyond 24 weeks provides confirmatory evidence.

METABOLIC AND CELLULAR EFFECTS OF PLACENTAL DYSFUNCTION

Oxygen and glucose consumption by the placenta is unaffected when nutrient delivery to the uterus is only mildly restricted and the fetal demands can be met by increased fractional extraction. Fetal hypoglycemia occurs uterine oxygen delivery and likely substrate delivery is less than a critical value and fetal oxygen uptake is reduced. Insulin is an important fetal growth factor. Fetal pancreatic insulin responses are blunted by mild hypoglycemia, allowing gluconeogenesis from hepatic glycogen stores. At this stage, fetal glucose stores and lactate are preferentially diverted to the placenta to maintain placental metabolic, endocrine, and nutrient transfer function . Hypoglycemia, hyper lactic acidemia , and growing base deficit correlate with the degree

of fetal hypoxemia and protein energy malnutrition. Down-regulation of several cellular transporters and the Na/H+ pump affects placental cellular function. Simultaneously, the principle endocrine growth axis(insulin and insulin like growth factors) as well as leptin-coordinated fat deposition is down-regulated.

FETAL RESPONSE IN MAJOR ORGANS

Enhanced blood flow to the individual organs is documented in the myocardium, spleen, and liver. Conversely, blood flow resistance in the peripheral pulmonary arteries, celiac axis, mesenteric vessels, kidneys, and femoral and iliac arteries increases. The overall effect is an improved distribution of well-oxygenated blood to vital organs, with preferential streaming of descending aorta blood flow to the placenta for reoxygenation. There is progressive decrease in the amniotic fluid volume after long-standing redistribution.

A delay occurs in all aspects of central nervous system maturation in fetuses with chronic hypoxemia. There is also a progressive decline in global fetal activity. This results in higher baseline heart rate, with lower short- and long-term variation.

FETAL DECOMPENSATION

If placental dysfunction is progressive or sustained, the adaptive mechanisms become exhausted and decompensation begins. Multiple-

organ failure as a result of placental dysfunction is caused by the metabolic milieu and the regulatory loss of cardiovascular hemostasis. Metabolic abnormalities are exaggerated , acidemia worsens, and the risk of intrauterine damage or perinatal death increase dramatically.

5. DIAGNOSIS OF FGR

Diagnosis of FGR is important because it has demonstrable effects on survival and development of fetus.

CLINICAL ASSESSMENT

Clinical assessment is a reasonable screening tool for FGR in low risk pregnancies. Clinical assessment is based on assessment of past and present risk factors, physical examination, and ultrasound studies.

Accurate assessment of gestational age — Accurate knowledge of gestational age (GA) is critical to the diagnosis of FGR, given that normal and abnormal fetal size are defined, in part, by comparing the fetal weight of the index fetus to that of other fetuses of the same gestational age.

Symphysis-fundal height measurement —Serial measurement of the distance between the upper edge of the pubic symphysis and the top of the uterine fundus using a tape measure is a simple, inexpensive, and widespread procedure performed during antenatal care to detect fetuses that are poorly grown. Between 18 and 30 weeks, the symphysis-fundal height in centimeters coincides within 2 weeks of gestational age. The

first suspicion of FGR often arises when this length is noted to be discordant with the expected size for dates, that is at least three centimeters below the GA in weeks (eg, fundal height 32 cm at 36 weeks of gestation) [(42)].

The accuracy of fundal height measurements for screening and diagnosis of FGR is controversial; Observational studies using symphysis-fundal height measurements have reported a wide range of sensitivities: 28 to 86 percent of small fetuses were detected [35-38].

Abdominal palpation — Clinical assessment of fetal size by abdominal palpation does not perform well as a test for detecting FGR: sensitivities range from 30 to 50 percent [38,39,44].

SONOGRAPHIC SCREENING AND DIAGNOSIS

An initial sonographic examination at 16-20weeks followed by a second examination at 32-34weeks serial sonography should serve to identify many cases of fetal growth restriction (Ewigman and colleagues,1993).

Commonly used parameters include biparietal diameter, head circumference, abdominal circumference, femur length and various morphometric ratios like HC/AC, and FL/AC. Ultrasound results need to be interpreted in terms of pretest risk of FGR and take into account

whether the subject population was at low, moderate, or high risk of fetal growth abnormality.

The morphometric tests are more likely to overlook fetuses with symmetric FGR, but can be used as confirmatory tests of suspected asymmetric FGR. As discussed above, symmetric FGR comprises 20 to 30 percent of growth restricted fetuses and asymmetric FGR occurs in the remaining 70 to 80 percent of the FGR population.

Abdominal circumference — When fetal growth is compromised, the fetal abdominal circumference (AC) is smaller than expected because of depletion of abdominal adipose tissue and decreased hepatic size related to reduced glycogen storage in the liver. An abdominal circumference within the normal range for gestational age reliably excludes growth restriction, whereas a measurement less than 5th percentile is highly suggestive of growth restriction (American College of Obstetricians and Gynecologists, 2000b).

Studies report that reduced AC is the most sensitive single morphometric indicator of FGR[40-43]. The performance of AC measurement was illustrated by a study of 3616 pregnancies over 25 weeks of gestation that had a single ultrasound examination performed within two weeks of delivery [45]. AC measurement predicted small for gestational age (SGA) infants (i.e., birth weight below the 10th percentile

for GA) with sensitivity, specificity, positive and negative predictive values of 61, 95, 86, and 83 percent, respectively.

Measurement of AC was more predictive of FGR than measurement of either head circumference (HC) or biparietal diameter (BPD) or the combination of AC with either one of these two variables. The optimal time to screen for FGR was at approximately 34 weeks of gestation.

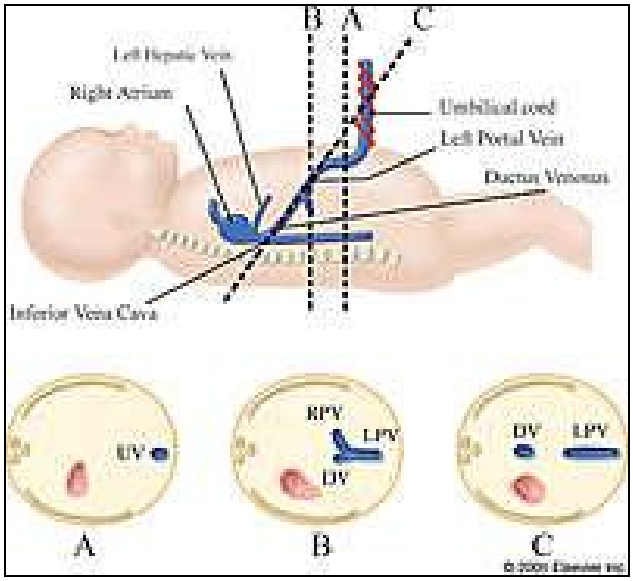
The following factors affect the sensitivity of the AC measurement:

- Symmetric versus asymmetric growth abnormality. AC is more sensitive in asymmetric FGR. [46].
- Gestational age. AC is more sensitive later in gestation. [47].
- Time interval between AC measurements. AC is more sensitive when the interval between measurements is more than two weeks[48].

MEASUREMENT OF ABDOMINAL CIRCUMFERENCE:

The abdominal circumference is obtained in the transaxial view of the fetal abdomen, at the level of fetal liver, using umbilical portion of the left portal vein as a landmark. The fetal stomach is at the same level, which is slightly caudal to the fetal heart and cephalad to the kidneys.

ABDOMINAL CIRCUMFERENCE



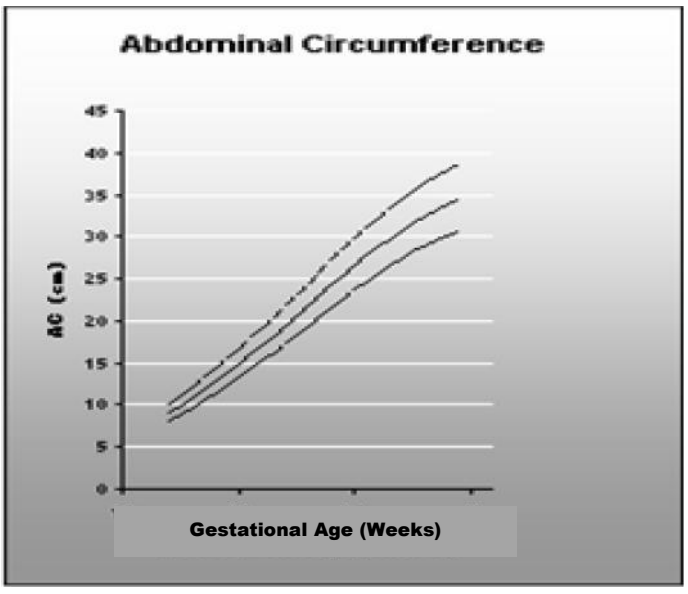
Umbilical venous circulation through the fetal liver. A. Plane of section depicting the umbilical vein (UV) in short axis.This plane is too caudal for abdominal circumference measurement. B. Plane of section through the junction of the left (LPV) and right (RPV) portal veins.This is the correct level for AC measurement (DV, ductus venosus). C. Plane of section aligned along the course of the LPV.Note that this plane is too inclined in a craniocaudal axis. (Illustration by James A. Cooper, MD, San Diego, CA.)

Plane of measuring abdominal circumference

Sonographic appearance of the cerebellum through gestation

Normal Range for Abdominal Circumference

Gestational age range (weeks + days)	Abdominal Circumference (mm)		
	5th centile	Median	95th centile
14+0-14+6	80	90	102
15+0-15+6	88	99	112
16+0-16+6	96	108	122
17+0-17+6	105	118	133
18+0-18+6	114	128	144
19+0-19+6	123	139	156
20+0-20+6	133	149	168
21+0-21+6	143	161	181
22+0-22+6	153	172	193
23+0-23+6	163	183	206
24+0-24+6	174	195	219
25+0-25+6	184	207	233
26+0-26+6	195	219	246
27+0-27+6	205	231	259
28+0-28+6	216	243	272
29+0-29+6	226	254	285
30+0-30+6	237	266	298
31+0-31+6	246	277	310
32+0-32+6	256	287	322
33+0-33+6	265	297	334
34+0-34+6	274	307	345
35+0-35+6	282	316	355
36+0-36+6	289	324	364
37+0-37+6	295	332	372
38+0-38+6	302	339	380
39+0-39+6	307	345	387



Estimated fetal weight (EFW) : Fetal weight estimation has become one of the most common methods of identifying the growth-restricted fetus. Equations that incorporate AC, BPD, and FL seem to provide the most accurate estimates of fetal weight[49]. In general, estimated fetal weight measurements are within 10 percent of the actual birthweight in 75 percent of patients in whom there is a clinical suspicion of FGR.

The average sensitivity, specificity, positive and negative predictive values for FGR using these parameter are approximately 90, 85, 80, and 90 percent, respectively [55-58]. The sensitivity is generally higher for infants with severe growth restriction (birth weight less than the 3rd percentile). But this can diagnose FGR only when the gestational age is known.

Customized growth curves — EFW is typically classified using population-based birth weight centiles. Several studies have compared the use of population-based birth weight centiles to customized centiles for prediction of SGA and perinatal morbidity. These studies have generally concluded that using a customized birth weight standard increases the identification of fetuses at risk of perinatal death and neonatal morbidity. This improvement in prediction of outcome may be related to better identification of the constitutionally small fetus through adjustment for maternal characteristics, or to use of an intrauterine (ultrasound) growth

BIRTH WEIGHT PERCENTILE

Age (wk)	5th	10th	50th	90th	95th
20	249	275	412	772	912
21	280	314	433	790	957
22	330	376	496	826	1023
23	385	440	582	882	1107
24	435	498	674	977	1223
25	480	558	779	1138	1397
26	529	625	899	1362	1640
27	591	702	1035	1635	1927
28	670	798	1196	1977	2237
29	772	925	1394	2361	2553
30	910	1085	1637	2710	2847
31	1088	1278	1918	2986	3108
32	1294	1495	2203	3200	3338
33	1513	1725	2458	3370	3536
34	1735	1950	2667	3502	3697
35	1950	2159	2831	3596	3812
36	2156	2354	2974	3668	3888
37	2357	2541	3117	3755	3956
38	2543	2714	3263	3867	4027
39	2685	2852	3400	3980	4107
40	2761	2929	3495	4060	4185
41	2777	2948	3527	4094	4217
42	2764	2935	3522	4098	4213
43	2741	2907	3505	4096	4178
44	2724	2885	3491	4096	4122

Source : Alexander and associates (1996).

standard rather than a birth weight standard for classification of FGR. Since fetuses who are born preterm tend to have lower birth weights that fetuses of the same gestational age who remain inutero, using an intrauterine weight standard increases identification of FGR remote from term.

Growth velocity — As discussed above, the use of any parameter (eg, AC, EFW) in the prediction of FGR is based upon accurate assessment of GA. If dates are unknown, serial sonographic examinations at two-week intervals should be performed to evaluate the rate of interval growth (ie, growth velocity). Irrespective of GA, there is a significantly lower rate of change over time of AC or EFW in FGR fetuses compared with those fetuses whose growth is appropriate for GA . In one study, as an example, a change in fetal AC of less than 10 mm over a two-week period had a sensitivity of 85 percent and specificity of 74 percent for identifying FGR[50]. Fetuses with normal growth velocity are at low risk of complications associated with FGR.

Body proportions — The HC/AC ratio, FL/AC ratio, and ponderal index have also been used to identify the growth restricted fetus, particularly in the setting of asymmetric FGR.

HC/AC ratio — The HC/AC ratio has been proposed for evaluating fetuses with asymmetric FGR. In these infants, the size of the liver tends

to be disproportionately small compared to the circumference of the head or length of the femur, which are initially spared from the effects of nutritional deficiency.

The HC/AC ratio decreases linearly throughout pregnancy and a ratio greater than 2 standard deviations (SD) above the mean for GA is considered abnormal. The sensitivity, specificity, positive and negative predictive values of an abnormal HC/AC in a population with FGR of mixed etiologies were 36, 90, 67, and 72 percent, respectively [51]. These findings demonstrate that an abnormal HC/AC ratio is more accurate in predicting FGR related to uteroplacental insufficiency (often asymmetric) than FGR from other etiologies (often symmetric). However, not all fetuses with an elevated HC/AC ratio have FGR. As an example, macrocephaly could also be associated with an abnormal HC/AC, which would be unrelated to FGR.

FL/AC ratio — The FL/AC ratio uses sonographic elements that relate to both weight and length in the prediction of FGR. An FL/AC ratio greater than 23.5 percent has a sensitivity of 56 to 64 percent and specificity of 74 to 90 percent for identification of asymmetric FGR[52]. This ratio is independent of GA in normally grown fetuses in the last half of pregnancy. However, an abnormal FL/AC ratio does not accurately diagnose symmetric FGR. The sensitivity, specificity, positive and

negative predictive values of the 90th percentile of FL/AC ratio in a mixed population of FGR fetuses were 30, 91,14, and 96 percent, respectively[53].

Therefore, the FL/AC ratio is unsuitable for screening for FGR in the general population.

Ponderal index:

PI is often used as an index (ie, PI = [weight (in g) x 100] ÷ [length (in cm)](3) to define growth restriction(54). A fetal PI has been calculated based upon a sonographically derived EFW and measurement of the FL. One study reported sensitivity, specificity, and positive predictive value of the fetal PI for FGR of 77, 82, and 36 percent, respectively; however, there was a poor correlation between fetal and neonatal PI [55].

With normal growth, the PI increases gradually from 30 to 37 weeks gestation and then remains constant. Decreased growth of adipose tissue and skeletal muscle, the major contributors to body weight, results in a reduced PI. Reductions in PI or other indices, such as the ratio of mid-arm to occipito-frontal circumference, can identify growth restriction in newborns whose weight is greater than the 10th percentile. PI of less than 10th percentile reflects fetal malnutrition; PI of less than third percentile indicates severe wasting .

Amniotic fluid volume — Oligohydramnios refers to amniotic fluid volume that is less than expected for gestational age. It is typically diagnosed by ultrasound examination and may be described qualitatively or quantitatively by various methods.

Oligohydramnios is one of the sequelae of FGR. The proposed mechanism is diminished fetal urine production due to hypoxia-induced redistribution of blood flow to vital organs at the expense of less vital organs, such as the kidney[56]. Oligohydramnios commonly occurs with complications of pregnancy other than FGR. In addition, a significant proportion (approximately 15 to 80 percent) of fetuses with FGR do not have decreased amniotic fluid volume. Therefore, oligohydramnios is a poor screening modality for suboptimal growth [43,57]. However, if it is present in the absence of ruptured membranes, congenital genitourinary anomalies, or prolonged pregnancy, FGR is the most likely etiology.

Soft tissue measurements — FGR results in a decrease in both adipose tissue and muscle mass. Measurement of fetal soft tissue is probably predictive of FGR; however, there are inadequate data for defining the best site for measurement or the sensitivity and specificity of this parameter.

Doppler velocimetry — Doppler ultrasound is a noninvasive technique commonly used to evaluate maternal and fetal hemodynamics.

Continuous, adequate perfusion of the maternal and fetal sides of the placenta is necessary for normal fetal growth. FGR is associated with diminished flow and abnormal Doppler waveforms in both maternal and fetal vessels. Doppler has been useful in evaluation and management of pregnancies suspected of FGR. Not all infants whose birth weight is below the 10th percentile have been exposed to a pathological process. Most small newborns are constitutionally small and healthy. Differentiating the fetus with pathological growth restriction who is at risk for perinatal complications from the constitutionally small, but healthy, fetus has been an ongoing challenge in obstetrics. This is the setting in which Doppler is useful because it can distinguish between these two groups[58,59] and guide timing of interventions (eg, intensive monitoring, antenatal glucocorticoids, early delivery) that reduce perinatal mortality.

Venous Doppler assessment has been studied less extensively, and is used for monitoring, rather than diagnosis, of FGR.

Uterine artery — Uterine artery Doppler flow velocimetry has limited diagnostic accuracy for prediction of FGR and is not useful as a screening tool.

The shape of the uterine artery velocity waveform is unique: it is characterized by high end-diastolic velocities with continuous forward

blood flow throughout diastole. As GA advances, the degree of end-diastolic flow typically increases; however, failure of normal endovascular trophoblastic invasion of the spiral arteries results in increased uterine artery vascular resistance and decreased perfusion of the placenta . Preeclampsia and/or FGR often subsequently develop [60].

The systolic/diastolic (S/D) ratio of the uterine artery in normal pregnancies should be less than 2.7 after the 26th week of gestation. If the end-diastolic flow does not increase throughout pregnancy or a small uterine artery notch is detected at the end of systole, the fetus is at high risk for developing FGR. Diastolic blood flow may be absent or even reversed with extreme degrees of placental dysfunction. Such findings are ominous and may precede fetal death or signal a high risk of abnormal fetal neurologic outcome.

Umbilical artery — Doppler umbilical artery studies are not useful for screening and diagnosis of FGR. Comparative studies are scarce, but support this conclusion.

Fetal cerebral arteries — Doppler evaluation of fetal cerebral arteries is not useful in the initial diagnosis of FGR, but may be useful confirming the diagnosis and evaluating the FGR fetus. In the normally developing fetus, the brain is an area of low vascular impedance and is the recipient of continuous forward flow throughout the cardiac cycle. Asymmetric

FGR is likely caused by redistribution of fetal blood flow to the fetal brain, at the expense of less essential areas such as subcutaneous tissue, kidneys, and liver. However, the already low cerebral resistance has to drop even further to enhance cerebral blood flow. This has been measured as increased end diastolic velocities and decreased S/D ratios in the cerebral arteries of growth restricted fetuses[62].

Venous Doppler — Venous Doppler has no role in diagnosis of FGR since venous Doppler abnormalities constitute a late circulatory finding. Venous Doppler assessment is used for monitoring fetal well-being; use of this technique appears to significantly improve the prediction of stillbirth and acidemia over use of umbilical artery Doppler alone.

6. THE NORMAL CEREBELLUM [63,64]

Cerebellum is located in the posterior fossa and consists of two hemispheres connected by the vermis. Cerebellum is peanut shaped with central constriction denoting the vermis and flared ends representing two hemispheres. Its location in the posterior fossa (surrounded by the dense petrous ridges and occipital bone) makes it more resistant to deformation by extrinsic pressure. It has therefore been proposed that the transverse cerebellar diameter is a better predictor predictor of gestational age than the BPD when there are variations in the shape of the fetal head (dolicocephaly or brachycephaly). On ultrasound, the cerebellar

hemispheres are normally echo-poor to moderately echogenic, bounded superiorly by the echogenic tentorium cerebella. Cistern magna is a fluid collection posterior to the cerebellum. The vermis separates the cisterna magna from the fourth ventricle.

Can be sonographically visualized as early as 9-10 weeks. It grows rapidly in the second trimester having a linear relationship with gestational age. Measurement in mm approximately equals the gestational age in weeks.

In prenatal ultrasound , an axial plane 15 to 30 degrees from the canthomeatal line visualizes both the cerebellum and cistern magna. This plane is usually reached by starting with the level where the standard BPD is obtained, then exaggerating the posterior tilt of the transducer to include the cerebellum. Measurement of the nuchal skin can also performed at this level in the early second trimester. The “banana sign” in fetuses with chiari 2 malformations is also seen at this level. Where the cerebellar hemispheres become oriented anteriorly and appear to wrap around the cerebral peduncles giving rise to the elongated crescentic “banana”.

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GRADES OF CEREBELLUM

Grade 1:

- Seen predominantly upto 27 weeks of gestation.
- Cerebellar hemisphere is rounded and lacks echogenicity.
- Vermis poorly developed giving the cerebellum the appearance of an “eyeglass”.

Grade 2:

- Seen predominantly from 28-32 weeks of gestation .
- Vermis more prominent and appears as an echogenic rectangular tissue connecting both hemispheres.
- Cerebellar hemisphere is oval and the central portion is more echogenic than the peduncles but less echogenic than the circumferential margin of the hemisphere.
- Cerebellum has “dumbbell” appearance

Grade 3:

- Seen predominantly after 32 weeks of gestation.
- Hemispheres become triangular or “fan-shaped”.
- Echo pattern from the central portion of the hemisphere is now similar to the margin of the vermis.
- Cerebellum now looks more solid than cystic

Transverse Cerebellar Diameter

The cerebellum can be measured in an axial plane using the transverse outer-to-outer margins. There is high degree of correlation between TCD and gestational age . Prior to 24 weeks the transverse cerebellar diameter in millimeters is equivalent to the gestational age in weeks following which there is a flattening of the growth curve[65].

Cerebellum is the last organ affected by decrease in the blood flow. In acute asphyxia, cerebellar blood flow remains unchanged as a consequence of redistribution of cardiac output[66]. To assess the fetal growth TCD has been one of the most reliable parameters in assessing the growth and gestational age estimation[67]. Thus TCD may serve as an independent indicator of GA against which other potential deviations of growth may be compared.

MEASUREMENT OF TRANSVERSE CEREBELLAR DIAMETER:

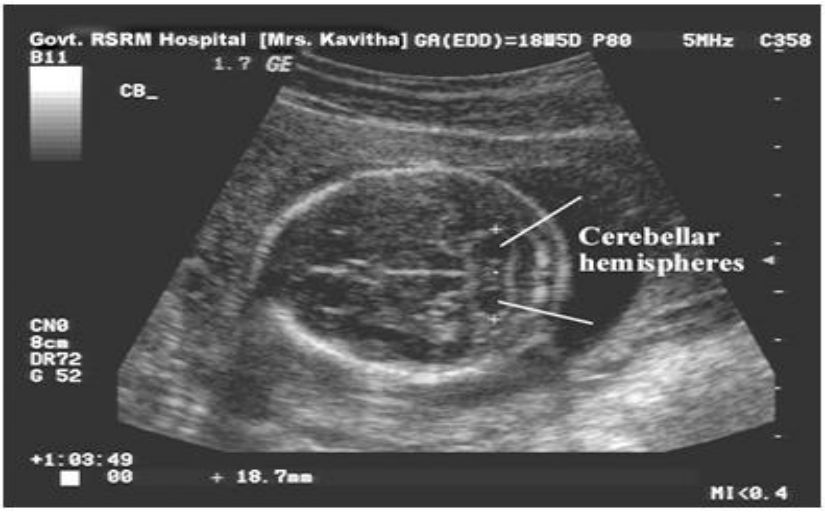
McLeasy et al (1984) and Goldenstein et al (1987) described the technique for measuring TCD, in which the usual thalamic plane used for BPD is obtained, the transducer is then rotated about 30^0 from reids baseline demonstrated the contents of posterior fossa. In all cases, the widest diameter of the cerebellum was measured.

The vermis of the cerebellum, cerebellar hemispheres, cisterna magna and the nuchal translucency are seen in this plane. The cerebral peduncles, the falx cerebri and the cavum septum pellucidi are imaged in the midline.

PREDICTED MENSTRUAL AGE ACCORDING TO TRANSVERSE CEREBELLAR DIAMETER MEASUREMENTS

Cerebellum (mm)	Menstrual Age (Week)	Cerebellum (mm)	Menstrual Age (Week)
14	15.2	35	29.4
15	15.8	36	30.0
16	16.5	37	30.6
17	17.2	38	31.2
18	17.9	39	31.8
19	18.6	40	32.3
20	19.3	41	32.8
21	20.0	42	33.4
22	20.7	43	33.9
23	21.4	44	34.4
24	22.1	45	34.8
25	22.8	46	35.3
26	23.5	47	35.7
27	24.2	48	36.1
28	24.9	49	36.5
29	25.5	50	36.8
30	26.2	51	37.2
31	26.9	52	37.5
32	27.5	53	38.0
33	28.1	54	38.3
34	28.8	55	38.5

Plane of measuring transverse cerebellar diameter



TCD/AC RATIO

This ratio compares the most preserved organ in the malnourished fetus, the cerebellum with the most compromised organ, liver, represented by fetal AC. In normally grown fetuses, there is a strong linear correlation with TCD measurement and AC. The TCD/AC ratio remains fairly constant throughout gestation. A value exceeding 2 SD of the mean was significantly associated with birth of small-for-gestational age infant, being abnormal in 98% and 71% of asymmetrically and symmetrically growth-retarded infants respectively[69].

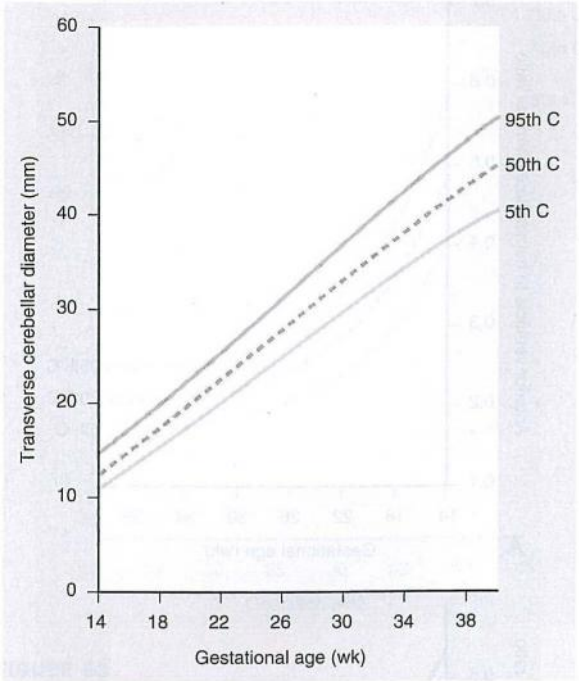
CALCULATION OF THE TCD/AC RATIO%:

TCD/ AC ratio% = TCD in cm /AC in cm x 100

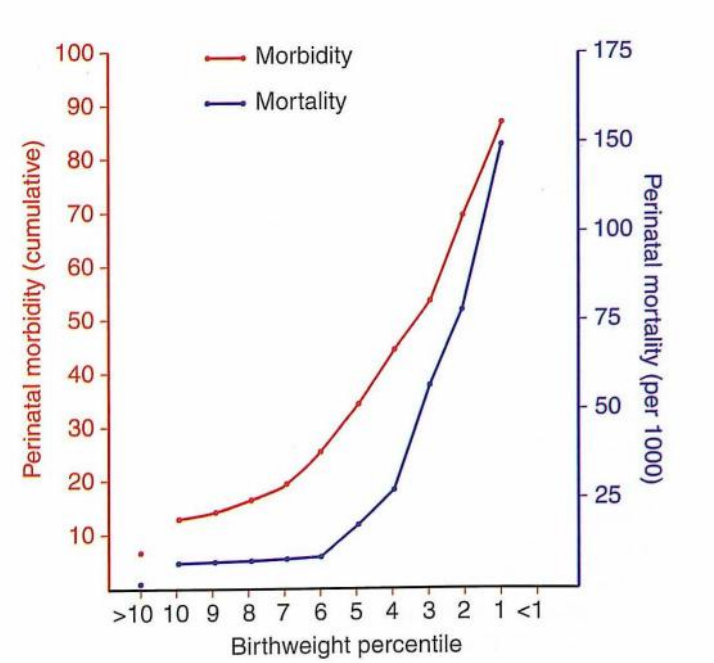
7. PERINATAL OUTCOME

Fetal growth restriction is the second leading cause of perinatal morbidity and mortality, followed only by prematurity. In assessing

Centile Chart for TCD



Relationship between Birth weight percentile and perinatal mortality and morbidity in SGA



perinatal outcome by weight, infants who weigh less than 2,500 g (5 lb, 8 oz) at term have a perinatal mortality rate that is five to 30 times greater than that of infants whose birth weights are at the 50th percentile. The mortality rate is 70 to 100 times higher in infants who weigh less than 1,500 g (3 lb, 5 oz). Perinatal asphyxia involving multiple organ systems is one of the most significant problems in growth-restricted infants. Timely diagnosis and management of IUGR is one of the major achievements in contemporary obstetrics. If the growth-restricted fetus is identified and appropriate management instituted, perinatal mortality can be reduced, underscoring the need for assessment of fetal growth at each prenatal visit.

REVIEW OF CLINICAL STUDIES

Reece et al (1987) stated that transverse cerebellar diameter was unaffected by IUGR, thus this nomogram measurement may serve as an independent and reliable correlate of gestational age against which potential deviations of growth may be compared[70] .

Mikovic and Markovic et al (1989) studied the growth of the fetal cerebellum in normal pregnancy between 20 and 40 weeks and proposed that TCD can be practically applied in cases where it is difficult or impossible to measure BPD or in cases where it is unsuitable because of expressed moulding of head. Since the cerebellum is not liable to changes

in its form and size as it is protected very well in the calvarium. It was found that there was a good correlation between multiple growth parameters and TCD. They proposed that the potential importance of TCD in the diagnosis of fetal intrauterine growth restriction is based on the assumption that the cerebellum is not liable or at least not considered liable for growth restriction[71].

Campbell (1991) done a prospective study in 162 patients, and measurement of TCD and AC was obtained between 15 to 38 weeks of gestation. The ratio between the TCD and AC was calculated. The mean ratio was 13.7%. This ratio remained constant during gestation[72].

Guan et al in 1992 generated a nomogram for TCD with respect to gestational age and compared fetal TCD, BPD, HC,AC and FL measurements by ultrasound. Correlation coefficient between the birth weight and their parameters were studied and concluded that the function of the cerebellar diameter in the evaluation of fetal growth and development is better than any other parameter .When combined with the abdominal circumference, the TCD may help to differentiate the types of growth restricted fetuses.

Campbell (1994) measured TCD, BPD, HC, AC, and FL using Hadlock's method of measurement and calculated TCD/AC ratio. The biometric measurements of the IUGR fetuses were significantly smaller

than the non-IUGR fetuses. The TCD is the only measurement that has a significant difference in the fetuses with IUGR compared to those who were not growth retarded. The IUGR fetuses had a larger TCD/AC ratio, the ratio value was greater than 15.9%. The sensitivity and specificity was 71% and 77% respectively and PPV 79% and NPV 68% for identification of IUGR cases. However 57% cases missed had severe growth restriction and not very useful in such cases[74].

T Tongsong et al (1999) analysed 167 pregnancies with suspected IUGR. The prevalence of IUGR among study group was 51.5%. The best cut off value of the TCD/AC ratio for predicting IUGR was 15.4%, giving the sensitivity, specificity, PPV and NPV of 73.26%, 80.25%, 79.75%, and 73.86% respectively. Concluded that the sonographic fetal TCD/AC ratio as a gestational age independent method can be helpful in antenatal diagnosis of IUGR, especially in pregnancies with uncertain gestational age[75].

Dilmen et al (1996) prospectively studied 330 pregnant women and measurements of TCD, AC, BPD, HC and TCD/AC ratio and HC/AC ratio were obtained between 16 and 41 weeks of gestation. The measurement of TCD had a very close relation to the gestational age. The TCD/AC ratio was calculated and found to be 0.1436$\pm$ 0.0106 (SD) which remained fairly constant throughout pregnancy. The 5th and 95th

percentiles were 0.1279 and 0.1603. Ten of eleven fetuses with TCD/AC ratios exceeding 2 SD (0.1648) were found to have asymmetrical intrauterine growth retardation upon neonatal examination. The TCD/AC ratio is valuable in identifying babies with asymmetrical IUGR in patients with SGA[76].

Vinkesteijn et al (2000) conducted a retrospective cross sectional study of the normally developing fetus with TCD increasing with advanced gestational age. A gestational age related normal reference chart was produced for TCD and concluded that increased TCD/AC values were suspicious of fetal growth restriction. The perinatal mortality in growth restricted fetuses with a small cerebellum was was increased twofold over that of other fetuses[77].

Malik et al (2003) measured TCD, AC and calculated TCD/AC ratio in pregnant women. The TCD/AC ratio was found to be 0.14064$\pm$ 0.059(SD) which remain fairly constant throughout gestation and thus it is a gestational age independent parameter. TCD was found to be an accurate parameter and it showed 92% accuracy in predicting GA. TCD/AC ratio was found to be a good tool to diagnose asymmetric IUGR. It was almost 100% accurate in diagnosing asymmetric IUGR in those with ratio exceeding 2 SDs[68].

Materials and Methods

MATERIALS AND METHODS

A prospective study consisting of 100 antenatal women was conducted in Government RSRM Lying In Hospital, Stanley medical college, Chennai during the period from November 2010 to October 2011.

INCLUSION CRITERIA

- Antenatal women with singleton live intrauterine gestation
- Antenatal women with excellent dates

EXCLUSION CRITERIA

- Antenatal women with unreliable dates
- Antenatal women with fetal anomalies
- Antenatal women with multiple gestations

METHODS

Antenatal women were enrolled after written informed consent. A detailed history of the patients was taken. A thorough systemic and obstetric examination was made. All preliminary investigations were done. The antenatal women were made aware of the benefits of ultrasonogram. These women were offered ultrasonogram between 20-22 weeks of gestation. The scans were carried out by the trained sonologist. With ultrasonogram the transverse cerebellar diameter (TCD) and abdominal circumference (AC) of fetus were measured in addition to anomaly scanning, routine biometric parameters and liquor volume. The

TCD/AC ratio was calculated. These women were informed about the results of the scan. These women were informed to come between 32-34 weeks for repeat scan. The patients were followed up till delivery. All babies at birth were assessed by the neonatologist and grouped as appropriate for gestational age (AGA) or fetal growth restriction (FGR) according to birth weight 10^{th} -90^{th} percentile and <10^{th} percentile for gestational age respectively. The babies were typed as symmetric or asymmetric IUGR based on ponderal index. Apgar score, NICU admission in days and perinatal outcome were noted. The cut-off of value of TCD/AC ratio for diagnosing FGR arrived by finding mean$\pm$2SD of TCD/AC ratio of AGA fetuses.

STATISTICAL ANALYSIS:

Statistical analysis was performed with SPSS software (version 15.00 for windows). To find out the statistical significance, linear regression, one way ANOVA (analysis of variance) test and student 't' test was done. For the purpose of this study 95% confidence interval has been chosen and 'p' value <0.05 has been taken as significant.

INTERPRETATION

The TCD/AC ratio >2SD considered to be at risk of having fetal growth restriction. Women who had TCD/AC >2SD at 20-22 weeks are considered to be at risk of symmetric FGR and at 32-34 weeks for asymmetric FGR.

Results and Analysis

RESULTS AND ANALYSIS

TABLE1: MATERNAL AGE DISTRIBUTION

Maternal Age in Years	Number of Cases	Percentage
18-20	10	10%
21-25	57	57%
26-30	29	29%
>30	4	4%

Most of the antenatal women (57%) of antenatal women were in the age group of 21-25 years.

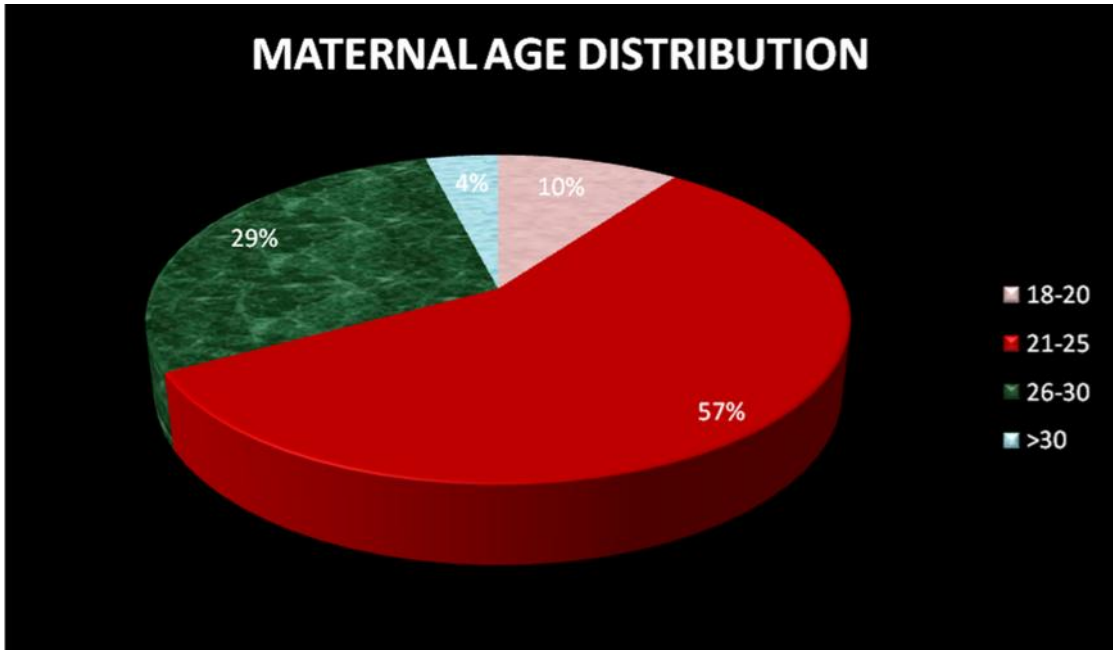


TABLE 2: PARITY

PARITY	NO OF CASES	PERCENTAGE
PRIMI	63	63%
MULTI	37	37%

63% of antenatal women were primigravida.

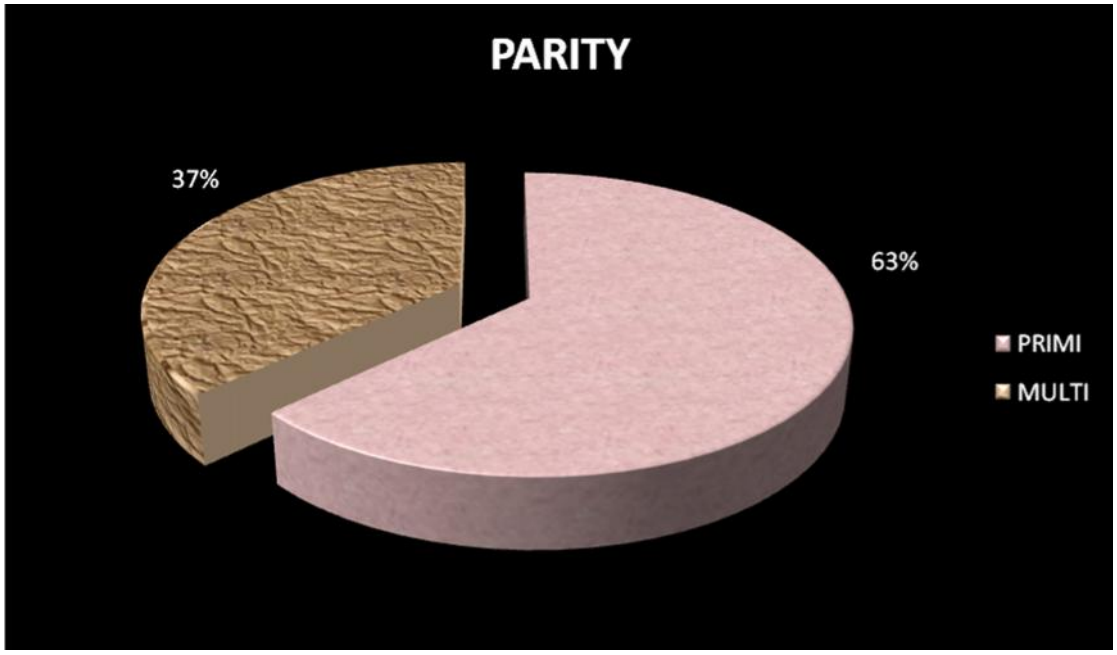


TABLE 3: RISK FACTORS

RISK FACTORS	NUMBER OF CASES	AGA		FGR	
		Number	Percentage	Number	Percentage
Preeclampsia	7	4	4.5%	3	30%
Oligohydramnios	3	1	1.1%	2	20%
Preeclampsia & Oligohydramnios	2	0	0%	2	20%
GDM	2	2	2.2%	0	0%
Chronic hypertension	1	0	0%	1	10%
No Risk Factors	85	83	92.2%	2	20%

30% of FGR were associated with preeclampsia, 20% with oligohydramnios and 20% with both risk factors.

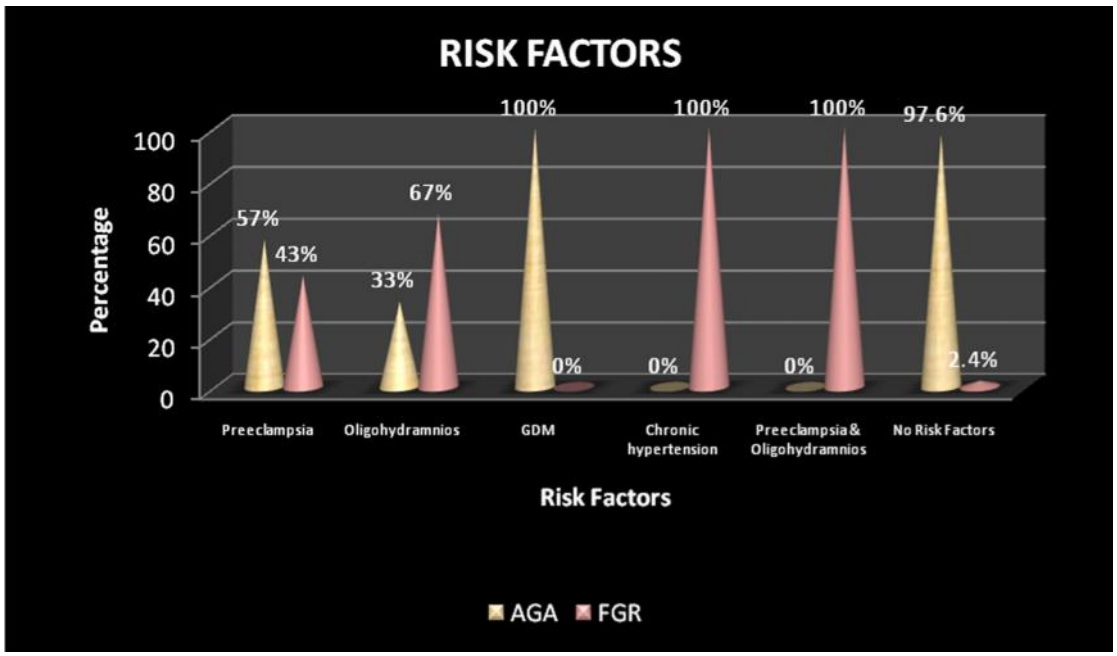


TABLE 4 : GESTATIONAL AGE AT DELIVERY

Gestational age at delivery in Weeks	Number of cases	AGA		FGR	
		Number	Percentage	Number	Percentage
34-36	6	4	4.4%	2	40%
37-40	94	86	95.6%	8	80%
Total	100	90	100%	10	100%

Preterm deliveries were high in FGR (40%) where as in AGA, it was only 4.4%.

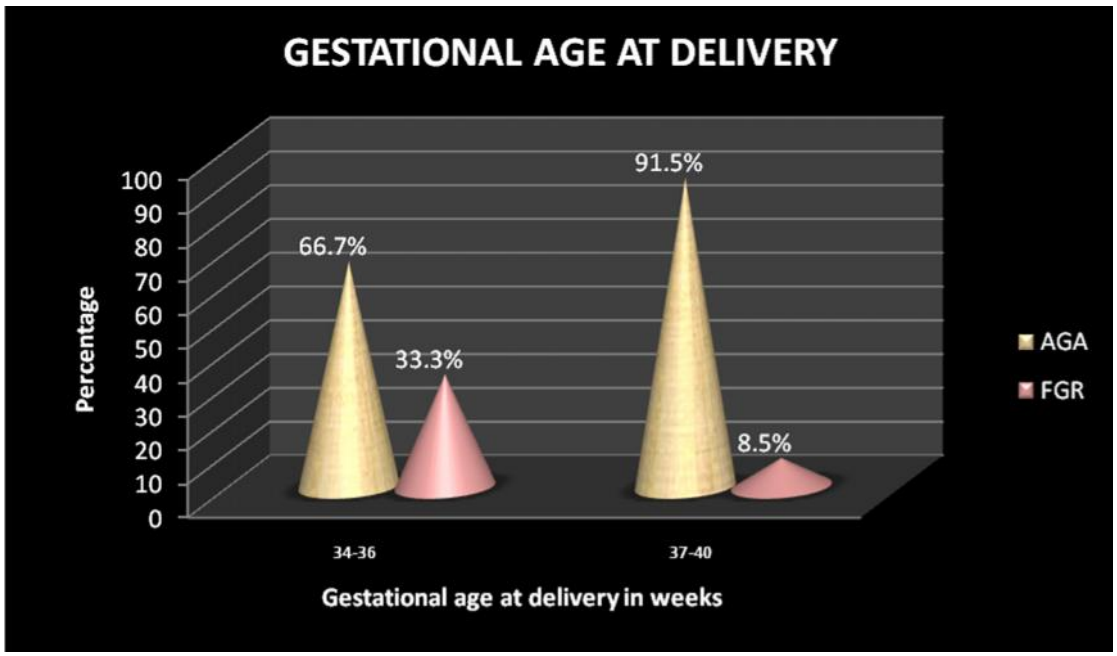


TABLE 5: MODE OF DELIVERY

Mode of Delivery	Number of cases	AGA		FGR	
		Number	Percentage	Number	Percentage
Vaginal	69	64	71%	5	50%
LSCS	31	26	29%	5	50%
Total	100	90	100%	10	100%

50% of pregnancies with Fetal Growth Restriction needed delivery by LSCS whereas only 29% of pregnancies with appropriate fetal growth needed LSCS.

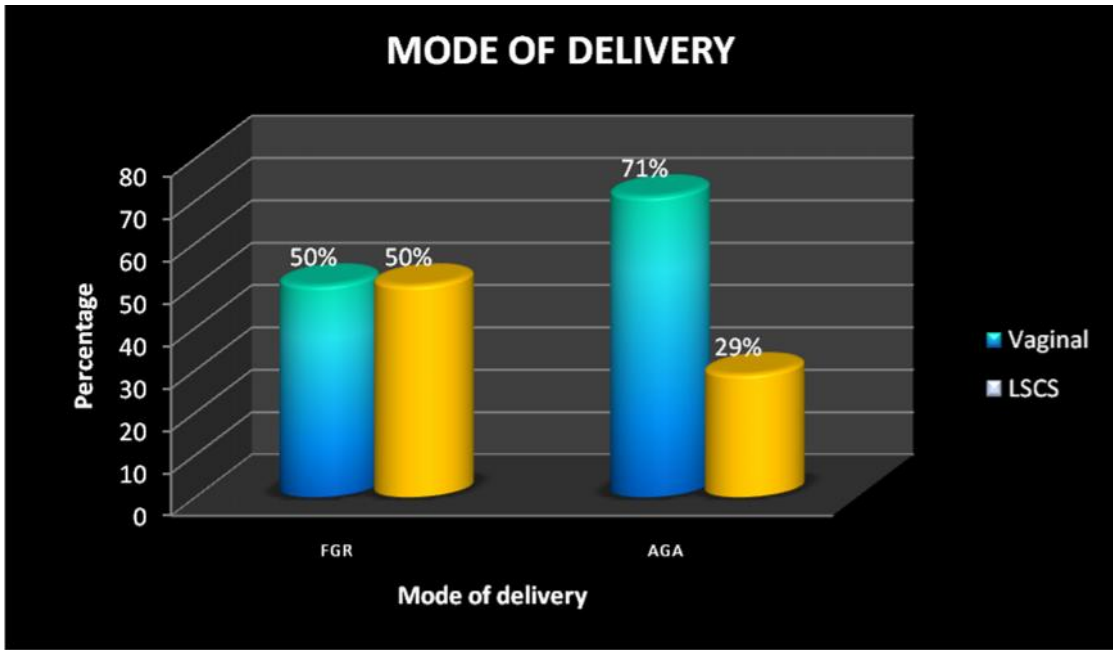


TABLE 6: IN UTERO GROWTH STATUS BASED ON PERCENTILE OF BIRTH WEIGHT

In utero growth Status	No of Cases	Percentage	Percentile
FGR	10	10%	$<10^{th}$
AGA	90	90%	$10\text{-}90^{th}$
LGA	0	0	$>90^{th}$

In our study, 10% of babies were FGR according to the birth weight percentile.

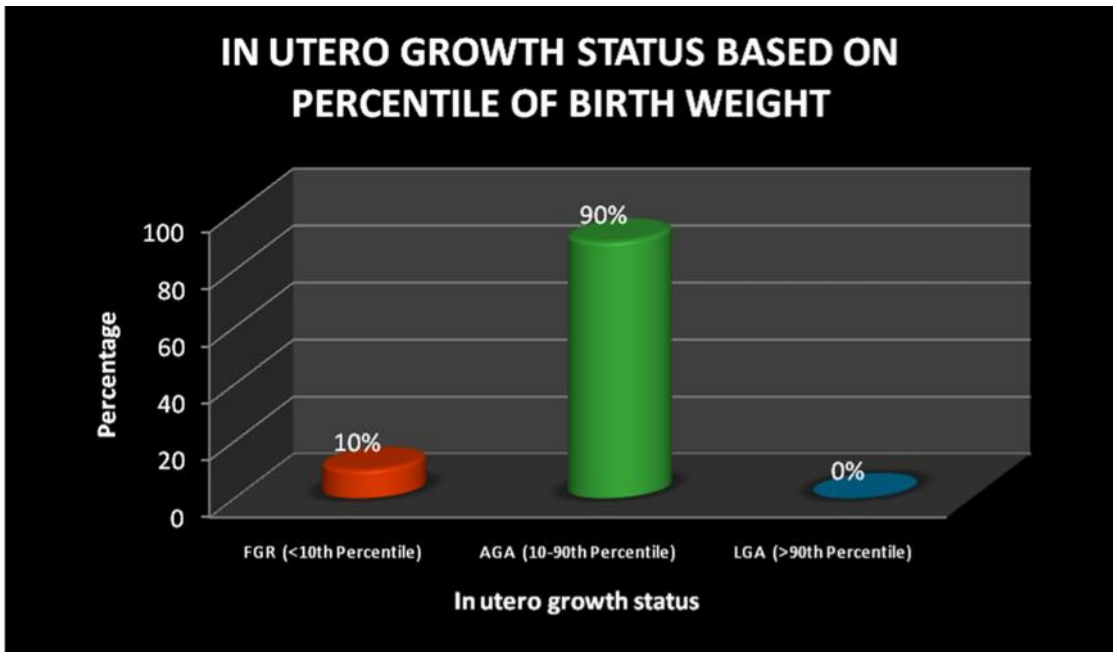


TABLE-7: CORRELATION OF GESTATIONAL AGE WITH TCD

GESTATIONAL AGE	CORRELATION COEFFICIENT	P value
20-22 WEEKS	r = 0.862	<0.001
32-34 WEEKS	r =0.803	<0.001

Significant correlation exists between Gestational Age and TCD.

TABLE-8 : CORRELATION OF GESTATIONAL AGE WITH AC

GESTATIONAL AGE	CORRELATION COEFFICIENT	P value
20-22 WEEKS	0.688	<0.001
32-34 WEEKS	0.486	<0.001

Significant correlation exists between gestational age and AC.

TABLE-9 : CORRELATION BETWEEN TCD AND AC AT 20-22 WEEKS

		TCD	AC
TCD	Pearson Correlation	1	.790(**)
	Sig. (2-tailed)	.	.000
	N	90	90
AC	Pearson Correlation	.790(**)	1
	Sig. (2-tailed)	.000	.
	N	90	90

** Correlation is significant at the 0.01 level (2-tailed).

TABLE-9 : CORRELATION BETWEEN TCD AND AC AT 32-34 WEEKS

		TCD	AC
TCD	Pearson Correlation	1	.569(**)
	Sig. (2-tailed)	.	.000
	N	90	90
AC	Pearson Correlation	.569(**)	1
	Sig. (2-tailed)	.000	.
	N	90	90

** Correlation is significant at the 0.01 level (2-tailed).

TABLE -10 : CORRELATION OF TCD & AC WITH IN UTERO GROWTH STATUS AT 32-34 WEEKS

Study parameters	In utero Growth Status				P value
	FGR		AGA		
	Mean	SD	Mean	SD	
TCD	3.96	0.18	4.03	0.15	0.183
AC	24.1	1.18	27.87	1.48	<0.001

There was no statistically significant difference between the mean TCD of FGR & AGA whereas there was statistically significant difference between the mean AC of FGR and AGA fetuses.

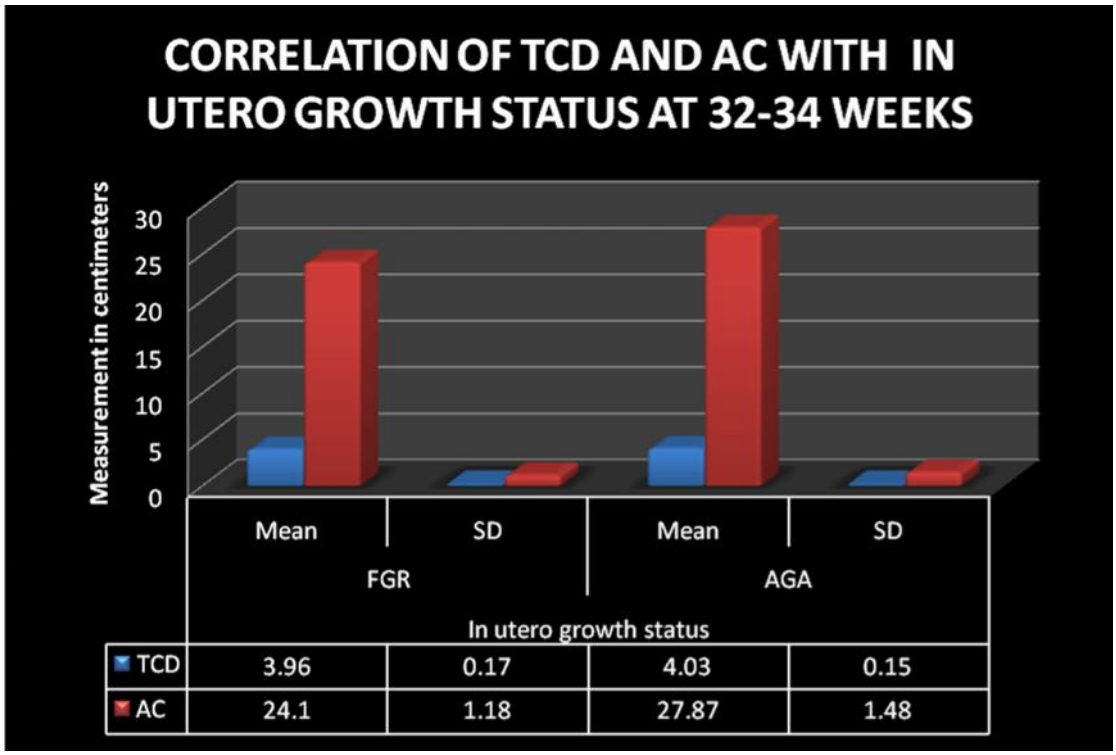


TABLE-11 : COMPARISON OF TCD/AC RATIO AT 20- 22 WEEKS AND 32-34 WEEKS IN AGA FETUSES

GA	TCD/AC RATIO	p VALUE
20-22 weeks	14.29±0.73(SD)	>0.073
32-34 weeks	14.49±0.62(SD)	

There was no statistically significant difference between the TCD/AC ratio at 20-22 weeks and 32-34weeks.

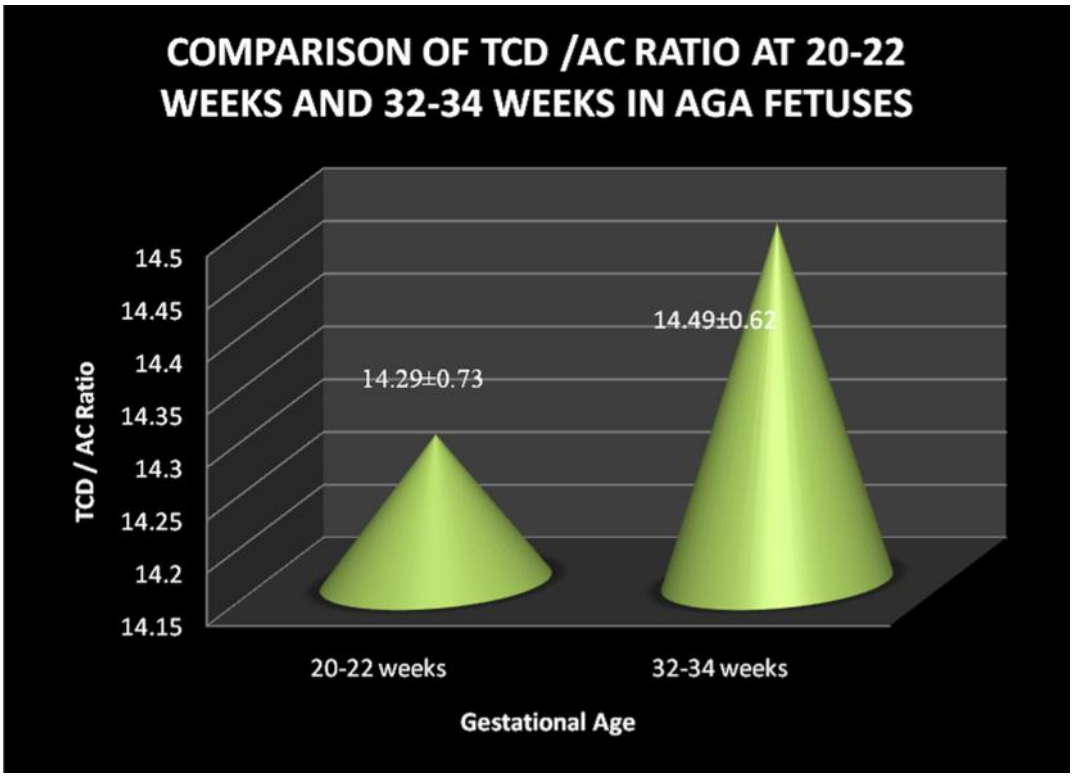


TABLE-12: DISTRIBUTION OF FGR/AGA FETUSES WITH RESPECT TO CUT-OFF VALUE OF TCD/AC RATIO AT 32-34 WEEKS

TCD/ AC Ratio 14.49+ 2SD (1.24)	FGR	AGA	TOTAL
≤ 15.73	2	89	91
> 15.73	8	1	9
Total	10	90	100

The cut-off value 15.73 diagnosed 8 cases of FGR and missed 2 cases of FGR.

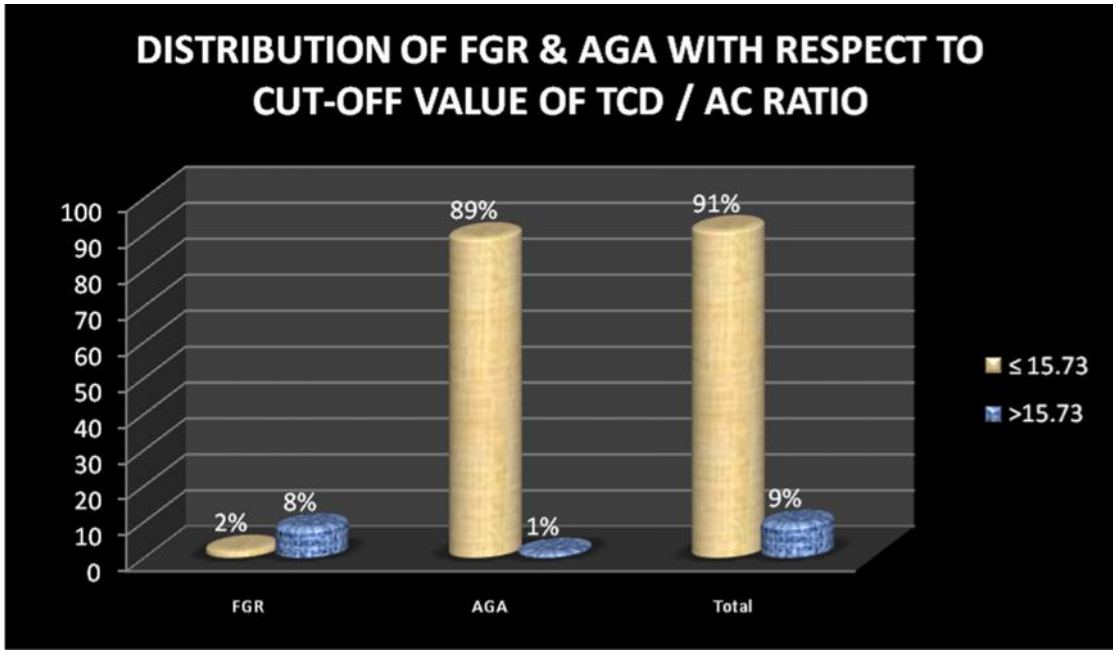


TABLE 13 : MEAN TCD/AC RATIO IN FGR AND AGA FETUSES AT 32-34 WEEKS

In utero growth status	TCD/AC Ratio	No of babies	p value
FGR	16.44	10	
AGA	14.49	90	<0.001

There was statistically significant difference between the mean TCD/AC ratio of FGR and AGA fetuses.

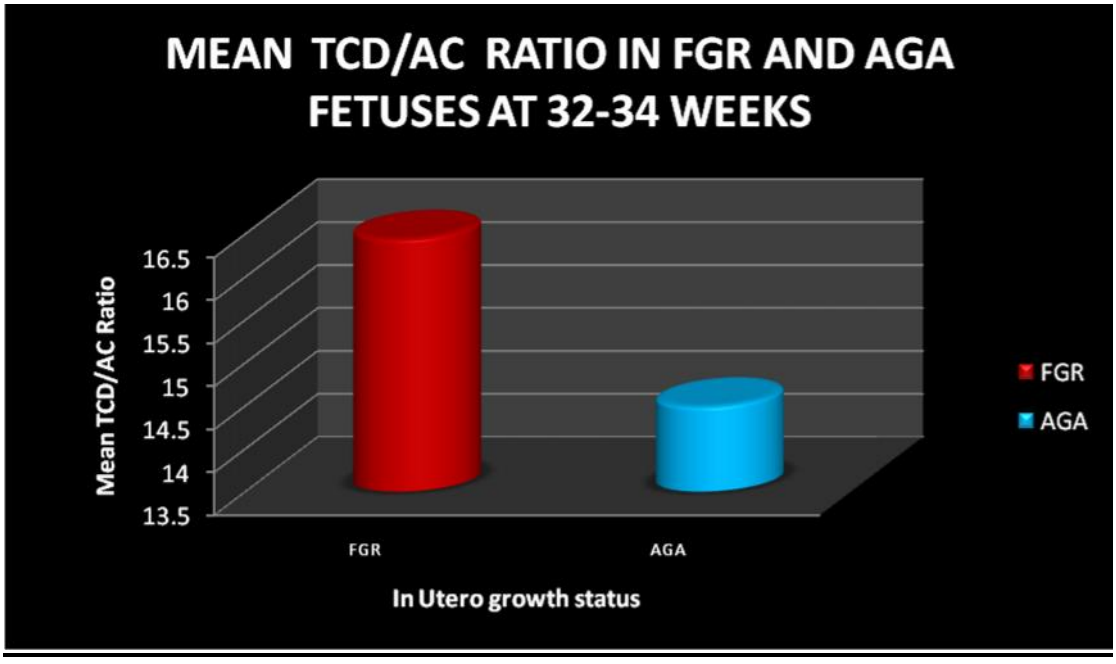


TABLE-14: DIAGNOSTIC VALUE OF TCD/AC RATIO IN FGR

Parameter	Sensitivity	Specificity	PPV	NPV
TCD/AC	80%	98.8%	88.8%	97.8%

Sensitivity and PPV of TCD/AC ratio in diagnosing FGR was 80% and 88.8% respectively. Specificity and NPV in diagnosing FGR was 98.8% and 97.8% respectively.

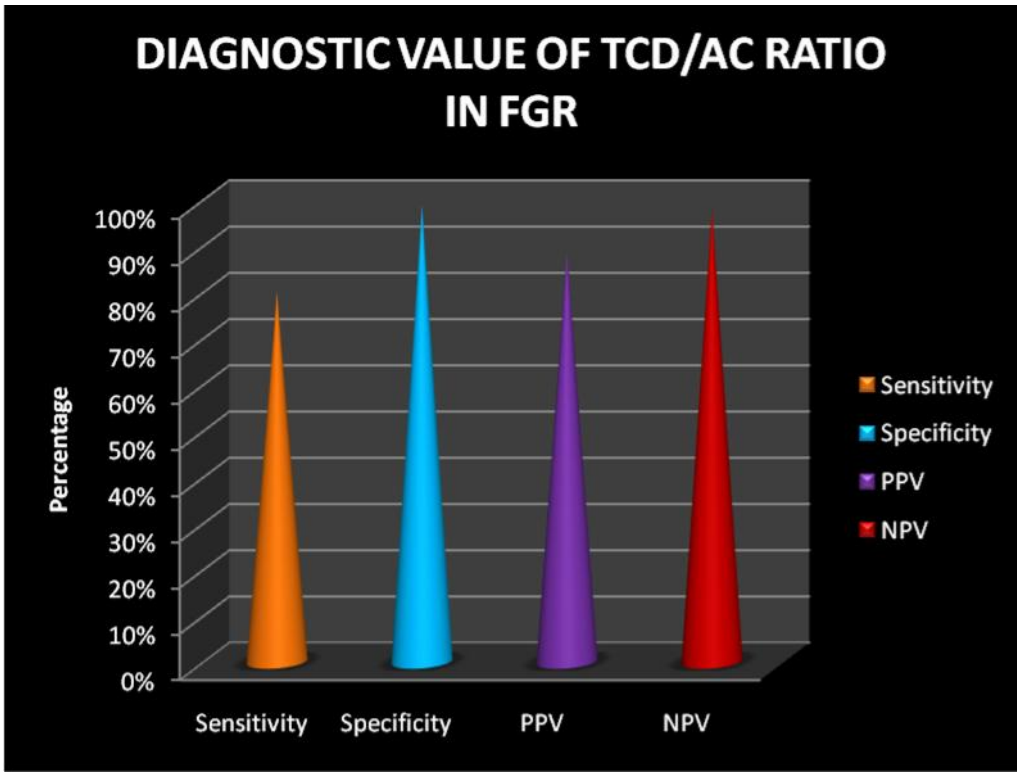
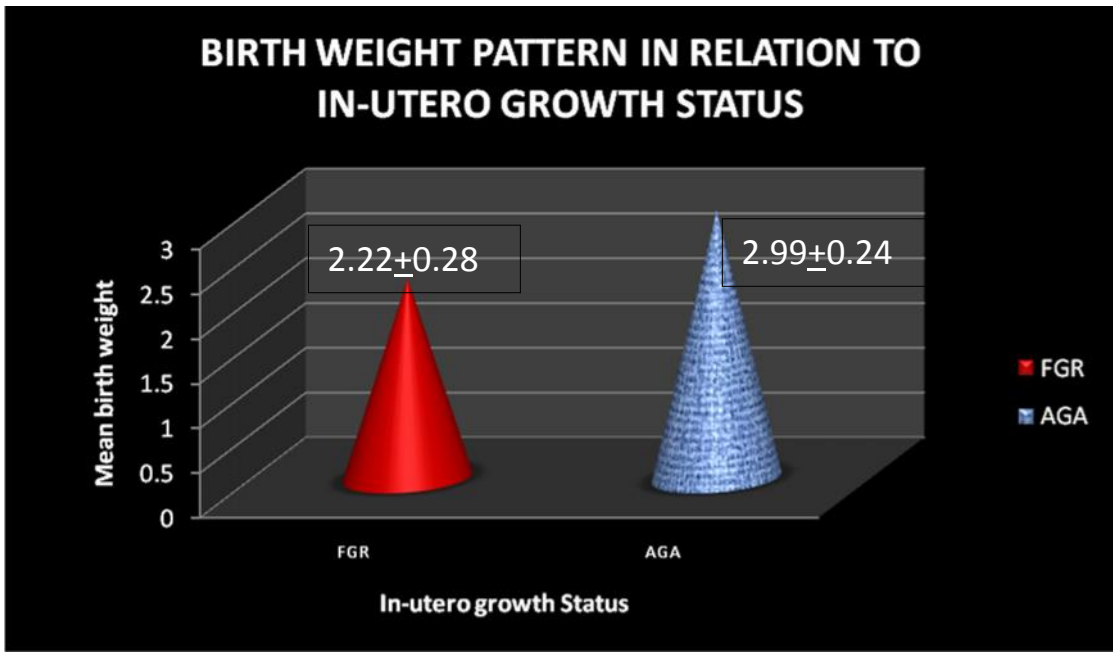


TABLE-15: BIRTH WEIGHT PATTERN IN RELATION TO IN-UTERO GROWTH STATUS

IN-UTERO GROWTH STATUS	BIRTH WEIGHT IN KG			P value
	Range	Mean	Mean ± SD	
FGR	1.5 – 2.25	2.22	2.22±0.28	**<0.001**
AGA	2.25 – 3.7	2.99	2.99±0.24	

Mean birth weight of babies with FGR is 2.22 kg. Mean birth weight of babies with AGA is 2.99 kg. There is statistically significant difference between the mean birthweight of both groups.



		Birth weight
TCD/AC at 20-22 weeks	Pearson Correlation	-.417(**)
	Sig. (2-tailed)	.000
	N	90
TCD/AC at 32-34 weeks	Pearson Correlation	-.544(**)
	Sig. (2-tailed)	.000
	N	90

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

TABLE-16 : APGAR SCORE

APGAR SCORE	SCORE < 7				SCORE ≥ 7			
	AGA		FGR		AGA		FGR	
	No.	%	No.	%	No.	%	No.	%
At 1 Minute	13	14.4%	9	90%	77	85.6%	1	10%
At 5 Minutes	1	1.1	3	30%	89	98.9%	7	70%

90% of FGR babies were scored 1 minute APGAR of <7 compared to only 14.4% in AGA.

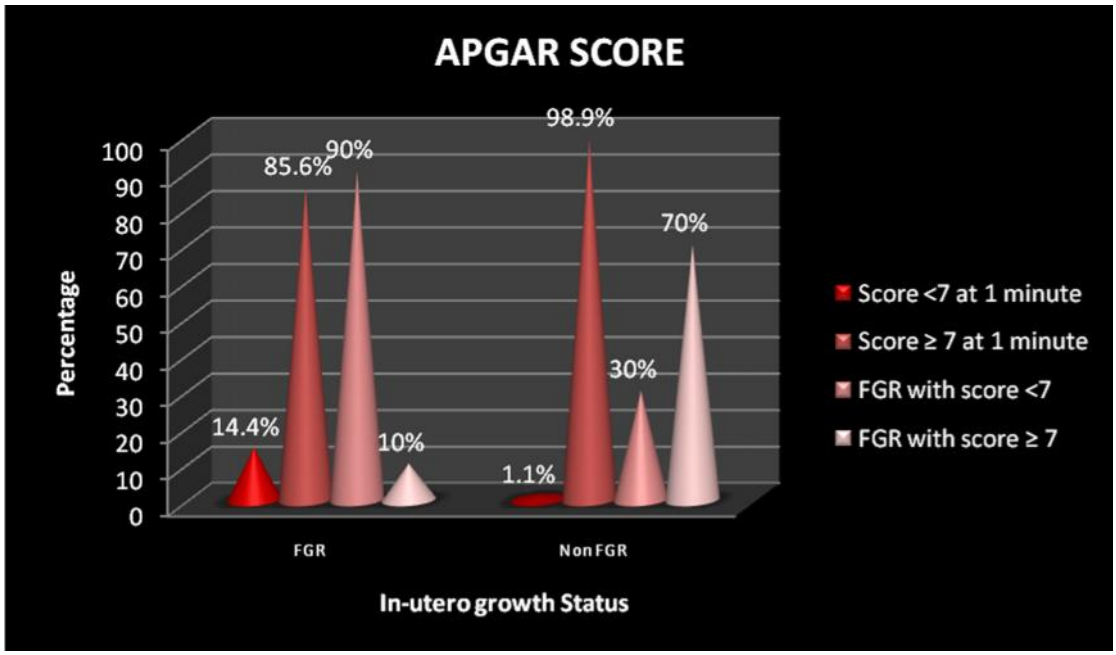


TABLE-16 : NICU ADMISSION

NICU ADMISSION IN DAYS	AGA		FGR	
	Number	Percentage	Number	Percentage
Nil	84	93.3%	1	10%
1-5	4	4.5%	6	60%
6-10	2	2.2%	2	20%
> 10	0	0%	1	10%

90% of FGR babies needed NICU admission whereas only 6.7% of the AGA babies needed NICU admission.

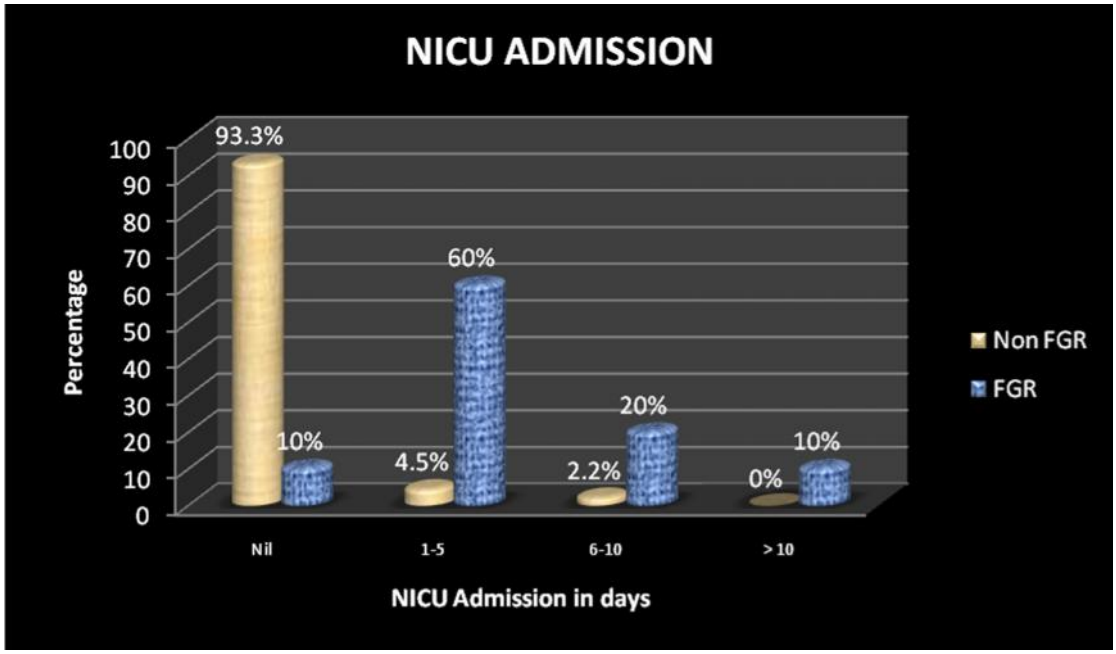


TABLE-17 : IN-UTERO GROWTH STATUS AND PERINATAL MORTALITY

IN-UTERO GROWTH STATUS	PERINATAL MORTALITY	
	Number	Percentage
AGA	0	0%
FGR	2	20%

The perinatal mortality was 20% among FGR babies whereas it was 0% among AGA babies.

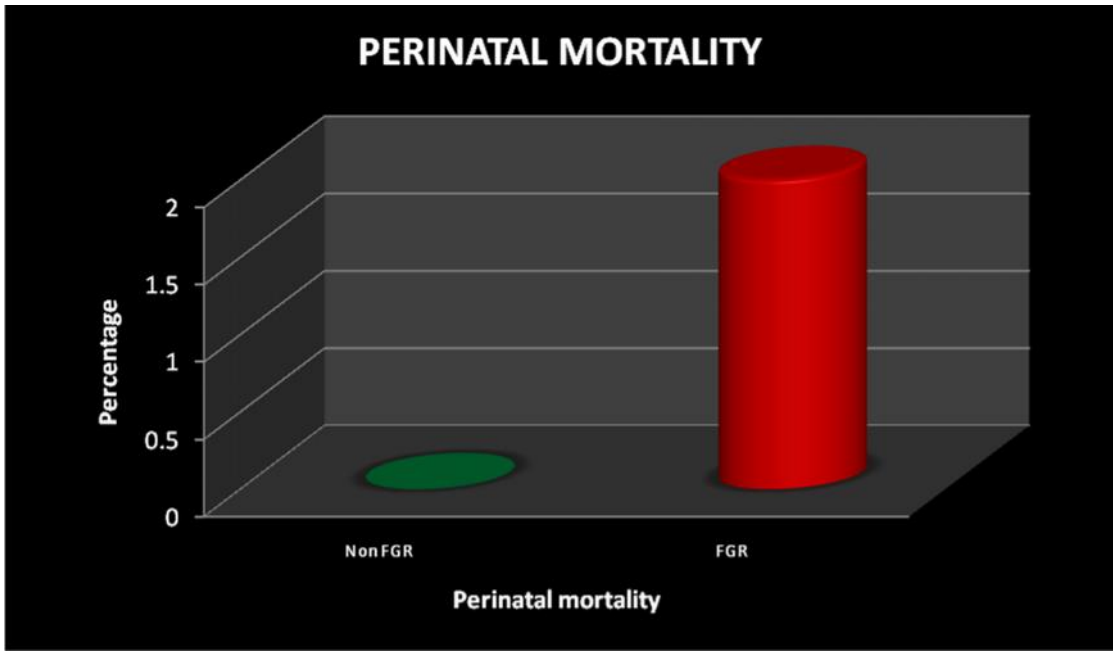
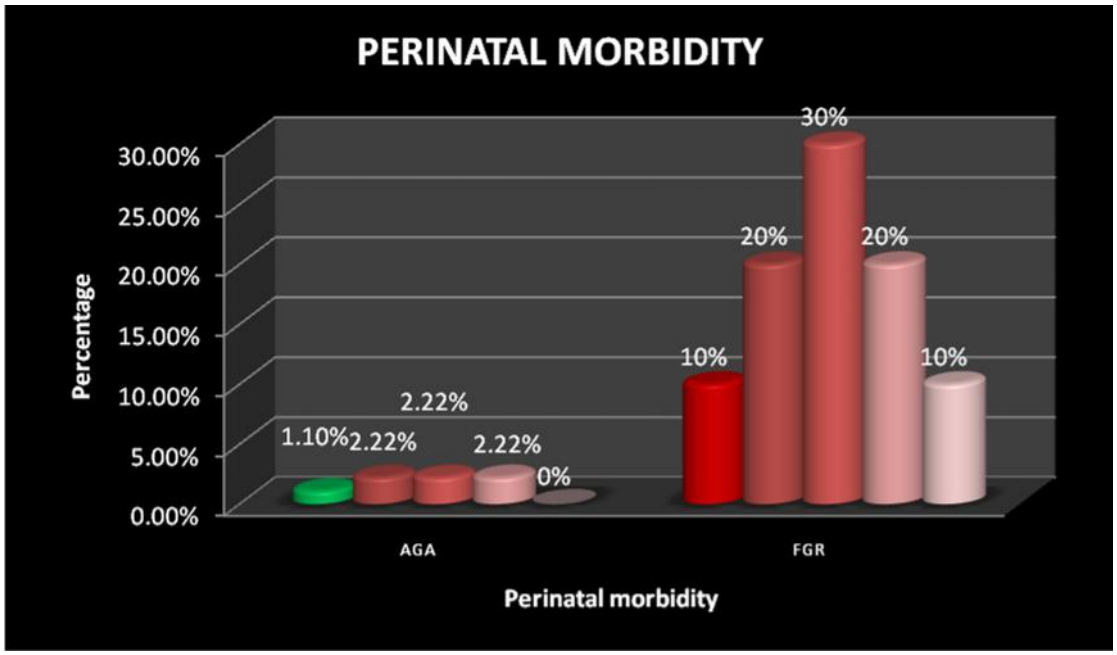


TABLE-18 : PERINATAL MORBIDITY

IN-UTERO GROWTH STATUS	AGA		FGR	
	Number	Percentage	Number	Percentage
Asphyxia	1	1.11%	1	10%
Meconium Aspiration Syndrome	2	2.22%	2	20%
Hypoglycemia	2	2.22%	3	30%
Hypocalcemia	2	2.22%	2	20%
Hypothermia	-		1	10%

Perinatal morbidity was high in FGR babies.



Discussion

DISCUSSION

In this study 100 antenatal women were selected after fulfilling the inclusion and exclusion criteria.

In the present study 57% of the women were between 21 to 25 Years and most of the women were primigravida accounted for 63%.

Risk factors associated with present pregnancy were preeclampsia in 9%, chronic hypertension in 1%, oligohydramnios in 5% and GDM in 2%. Of which 55.6% of preeclamptic and 80% of oligohydramnios patients delivered FGR babies. Patients with chronic hypertension and GDM also delivered FGR babies.

In our study 94% of patients were delivered at term and 6% of patients were delivered preterm. Out of 6 cases of preterm 33.3% were delivered FGR babies with TCD within mean$\pm$2SD of normal pregnancies.

In our study 69 women were delivered vaginally and 31 were delivered by LSCS. Among FGR 50% were delivered by LSCS, indications were fetal distress and oligohydramnios.

In the present study out of 100 pregnant women 10% were delivered FGR babies (birth weight $<10^{th}$ percentile), which is similar to general incidence of 3-10% [1].

In our study a strong correlation was noted between gestational age determined by last menstrual period and fetal TCD between 20-22 weeks(r = 0.876) and 32-34 weeks(r = 0.803) ($p<0.001$), similar to correlation of TCD and gestational age (r = 0.955) in the study conducted by Haller et al [78]. In the study conducted by Dilmen et al[76] the pearson correlation r =0.9767, which is also close to our study.

In our study a significant correlation exists between the gestational age determined by the last menstrual period and AC at 20-22 weeks (r = 0.688) and 32-34 weeks (r = 0.486) ($p<0.001$). In the study conducted by Haller et al [78] there was strong correlation exists between gestational age and AC (r = 0.9453) which is almost close to our study.

There was a significant linear correlation on noted between TCD and AC at 20-22 weeks (r = 0.790) and 32-34 weeks (r = 0.588) ($P<0.01$) in this study, similar to study conducted by Campbell et al [72] where strong linear correlation (r =0.918) noted between TCD and AC.

In our study there is no statistically significant difference between the mean TCD of FGR and AGA fetuses ($p>0.05$), Similar to study conducted by Reece et al[70], in which the TCD was not affected in FGR. In another study conducted by Vinkesteijn et al[76], the TCD was only mildly affected even in severe FGR, this is also close to our study. The concept that TCD is not affected even in FGR, is explained by the sparing

of blood flow to the cerebellum in IUGR as suggested by Berhman et al (1970)[3].

This study shows a statistically significant difference between the mean AC of FGR and AGA fetuses ($p<0.001$). This again proves that in FGR the AC will be affected more as the liver size is reduced due to reduced glycogen store[34].

In our study the TCD/AC ratio was 14.44±0.71 at 20-22 weeks (TCD1/AC1 ratio) and 14.49±0.62 at 32-34 weeks (TCD2/AC2 ratio) for normal pregnancies. There was no statistical difference between the two values implying that TCD/AC ratio was fairly constant throughout pregnancy, similar to study conducted by Meyer et al[69] , in which the TCD/AC ratio remained constant throughout pregnancy with respect to gestational age. In another study conducted by Campbell et al[72] ,the ratio calculated after each examination and the ratio remained constant which is also similar to our study.

In this study the mean $\pm$2SD of the TCD/AC ratio at 32-34 weeks is taken as cut-off value for diagnosing FGR. Here, the TCD/AC ratio is 14.49$\pm$0.62 comparable with study of Malik et al[68] in which the TCD/AC ratio was 14.06$\pm$0.59.

The cut-off value of TCD/AC ratio 15.73 in our study is close to the study conducted by meyer et al (1994)[69] in which the cut-off value for fetal growth restriction was15.9 ,and also with the study of haller et al [77]. In another study conducted by Tongsong et al(1999)[75] the cut-off value is 15.4 which is also close to our study.

In our study the sensitivity, specificity, PPV and NPV of TCD/AC ratio in diagnosing FGR were 80%, 98.8%, 88.8% and 97.8% respectively. Which is comparable with the study of Meyer et al(1994)[69] in which the sensitivity, specificity, PPV and NPV were 83.9%, 96.8%, 94.5% and 88.2% respectively.

In our study out of 10 cases of FGR 9 were asymmetrical and one symmetrical .out of 9 cases of asymmetric FGR 7 were diagnosed by the cut-off value of TCD/AC ratio with the sensitivity of 77.7%, similar to study of Meyer et al[69] where the sensitivity is 83.9%.

In our study one were symmetrical FGR which was diagnosed by the TCD/AC ratio with the sensitivity of 100%. In the study conducted by Meyer et al (1994)[69], TCD/AC ratio diagnosed symmetric FGR with a sensitivity of 71%. In our study the higher sensitivity is probably because of small sample size.

In our study one was found to be severe FGR which had TCD/AC ratio within cut-off value, and this is due to the fact that cerebellum mildly affected in severe FGR, similar to study by vinkesteijn et al[76].

In our study, significant negative correlation exists between the TCD/AC ratio and the birth weight.

Perinatal morbidity was high in FGR babies compared to AGA babies. Perinatal mortality was 20% in FGR babies.

Summary

SUMMARY

Hundred antenatal women with excellent dates attending antenatal clinic were selected in the department of obstetrics and gynaecology, R.S.R.M., Lying In hospital, Stanley medical college, Chennai.

Observations in the study includes

- Antenatal women in the study group were in 18-34 years with majority of the study population in the age group of 21-25 years.
- 63% of the patients in the study were primigravida and 37% were multigravida.
- Risk factors found in cases of fetal growth restriction were preeclampsia 50%, oligohydramnios 40%, and chronic hypertension in 10%.
- Ultrasonography was done twice between 20-22 weeks and 32-34 weeks and the TCD, AC measured and TCD/AC ratio calculated.
- Antenatal women were followed till delivery and the babies were examined by the neonatologist .Babies were grouped in to appropriate for gestational age(AGA), FGR according to birth weight percentile.
- In 100 antenatal women, 10 were delivered FGR babies.
- The ultrasonographic measurements TCD, AC and TCD/AC ratio were analyzed separately in both groups(women delivered AGA(90) or FGR(10)).

- Correlation were analyzed between gestational age and TCD, gestational age and AC and between TCD and AC. Strong correlation existed between these parameters in pregnancies with AGA fetuses.
- In pregnancies with FGR strong correlation existed between gestational age and TCD. Correlation were not significant between gestational age and AC, and TCD and AC as the reduced in FGR.
- The cut-off value of TCD/AC ratio for diagnosing FGR was arrived from mean±2SD of AGA fetuses at 32-34 weeks. The cut-off value in our study was 14.49+0.62.
- The 5^{th} and 95^{th} percentile of TCD/AC ratio in this study is 13.25-15.73 and the mean is 14.49±0.62(2SD) .
- The TCD/AC ratio was fairly constant throughout pregnancy.
- TCD/AC ratio diagnosed 8 out of 10 cases of FGR. The sensitivity, specificity, PPV and NPV 80%, 97.8%, 88.8% and98.8% respectively.
- TCD/AC ratio is a method used for assessment of fetal growth as it is independent of GA. There was a significant difference in the TCD/AC ratio of fetuses with or without FGR, thus providing a standard means of establishing growth parity.

Conclusion

CONCLUSION

1. The TCD and AC measurements correlates well with gestational age.
2. The TCD and AC has strong linear relationship, hence the TCD/AC ratio is fairly constant throughout pregnancy.
3. TCD unlike AC is not affected in FGR, because of brain sparing.
4. Hence, TCD/AC ratio is increased in FGR.
5. As the TCD/AC ratio is constant throughout the pregnancy, it is a gestational age independent parameter, can diagnose FGR in antenatal women with unknown gestational age.
6. Hence, TCD/AC ratio can be a screening test to diagnose FGR in the antenatal period. So, that early intervention could be attempted to improve the perinatal outcome.

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Annexures

Proforma

PROFORMA

Name

Age

Menstrual History

Obstetric Code

LMP

EDD

Dating Ultrasonogram : done / not done

Risk Factors

Preeclampsia	**Yes / No**
Chronic Hypertension	**Yes / No**
Oligohydramnios	**Yes / No**
Gestational Diabetes Mellitus	**Yes / No**
Chronic Renal Disease	**Yes / No**
Vasculopathy	**Yes/No**
Others	**Yes / No**

GENERAL PHYSICAL EXAMINATION

Built & nourishment

Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy / Edema

VITAL SIGNS : T: PR: RR: BP:

Breast :

Thyroid :

Spine :

HT : WT :

SYSTEMIC EXAMINATION

CVS :

RS :

CNS :

OBSTETRIC EXAMINATION

Date	**WT**	**BP**
P/A	**SFH**	**P/V**

INVESTIGATIONS

Routine:

Hb%	**HIV**	**BT**
PCV	**Hbs Ag**	**CT**
Urine Routine	**VDRL**	
Blood Group and RH Typing	**FBS**	**PPBS**
Blood Urea	**Serum Creatinine**	**Electrolytes**
Serum Uric acid	**Serum fibrinogen**	

LFT:

Total bilirubin:	**ALT:**	**AST:**
SAP:	**Total protein**	**Albumin:**

Urine:

Albumin	**Sugar:**	**Deposits**
SAP:	**Total protein:**	**Albumin:**

24 hours urinary protein:

Cardiotocography:

Ultrasonogram :

I Trimester USG:	
CRL:	
GSac	
GA	
FH	

USG at 20-22 weeks	
BPD	
HC	
FL	
AC	
TCD	
FH	
Liquor	
TCD/AC	
Anomaly	**Yes / No :**

USG at 32-34 weeks	
BPD	
HC	
FL	
AC	
TCD	
FH	
Liquor	
TCD/AC	

Gestational Age at Delivery :
Mode of Delivery: Vaginal / LSCS :
New born Details

- **Birth weight :**
- **In-utero growth status :**
- **APGAR at one minute :**
- **APGAR at 5 minutes :**
- **NICU Admission :**
- **Perinatal Morbidity :**
- **Perinatal Mortality :**

Master Chart

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
1	kala	26	primi	21	2.1	16.4	33.1	4	30.6	12.8	13.07	39		3.5	AGA		7	8	1			
2	nadhiya	24	primi	21.3	2.3	16.1	32.2	3.8	26.2	14.29	14.5	38		2.9	AGA		7	8	1			
3	viji	20	primi	21.5	2.4	18	33	4	29.2	13.33	13.7	39	3	3.3	AGA		7	8	2			
4	ameena	22	G2a1	20.2	1.9	13.6	32.2	3.8	27.5	14	13.82	39		2.9	AGA		7	8	1		1	
5	faritha	19	G2P1L1	21	2.3	15.1	33.2	4.2	27	15.23	15.55	37	1,2	2.25	FGR	3	4	7	1	1	2	
6	amudha	22	primi	21.1	2.3	16.4	32.3	3.9	27.2	14.02	14.34	38		2.7	AGA		7	8	1			
7	kanmani	24	primi	21.4	2.4	19.2	32.2	3.9	30.2	12.5	12.91	39		3.6	AGA		7	8	2			
8	sudha	29	G3A2	21.3	2.3	15.3	33	3.8	25.2	15.03	15.08	39		2.8	AGA		7	8	1			
9	dhivya	22	primi	22.2	2.5	18.4	34.2	4.2	30.7	13.59	13.68	38		3.3	AGA		7	8	2			
10	shakila	18	primi	21.2	2.2	14.3	33.5	4.1	27	15.38	15.19	39		3.3	AGA		7	8	2		2	
11	kamatchi	27	primi	20.1	2	15.2	32.4	3.9	28.1	13.16	13.88	39		3.2	AGA		7	8	1			
12	uma	21	primi	21.2	2.3	15	32.3	3.8	24.8	15.33	15.32	37		2.6	AGA		7	8	1		2	
13	nandhini	26	G2P1L1	21.1	2.2	15.3	33.1	4	27.2	14.38	14.71	38		2.75	AGA		6	8	1			
14	bharathy	28	primi	20.2	1.9	13.8	32.6	3.9	23.3	13.77	16.74	37	1,2	1.9	FGR	15	4	6	2	1	3	
15	priya	31	G3P1L1A1	21	2.1	16.4	32.2	3.9	30.6	12.8	12.75	39		3.7	AGA		7	8	1			

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
16	akila	20	primi	20.3	1.9	13.9	32.2	3.8	28	13.67	13.57	40		3.5	AGA		7	8	2			
17	vani	28	G2P1L1	21	2.1	14.6	33.3	4.2	28.5	14.38	14.74	36		2.6	AGA		7	8	1			
18	prema	22	primi	21.3	2.3	15.7	33.2	4.3	29.3	14.65	14.68	39		2.85	AGA		7	8	1			
19	nagamani	32	G2P1L1	21.1	2.2	15.3	32.3	3.6	23.5	14.38	15.32	37	4	1.5	FGR	10	7	8	1	1	1	1
20	parvathy	26	G2P1L1	21.4	2.3	16	33	4	27.3	14.38	14.65	38		2.8	AGA		7	8	1			
21	suryadevi	28	G3P2L1	20.5	2.1	14.3	32.2	3.9	23.5	14.69	16.6	38		2.2	FGR	2	5	7	1	1		
22	menaka	21	primi	21.5	2.4	16.9	34.1	4.3	29.6	14.2	14.53	39		2.95	AGA		7	8	2			
23	poongodi	28	G2A1	20.5	2.1	14	32.2	3.9	27	14.19	14.44	39		3.1	AGA		7	8	2			
24	chitra	24	G2P1L1	22	2.3	17	33.3	4	26.9	14.71	14.87	37		2.8	AGA		7	8	1			
25	abitha	26	primi	21.2	2.1	14.5	32.4	3.8	26.4	14.48	14.39	39		3.2	AGA		6	8	2			
26	ranjana	21	primi	22	2.3	16.4	32.6	4	27.6	14.02	14.49	39		2.9	AGA		7	8	2			
27	angel	23	primi	21.2	2.1	14.2	33.2	4.1	29	14.79	14.14	38	1	2.75	AGA		7	8	1			
28	selvi	20	primi	21.1	2.3	17.9	33.4	4.1	30.8	12.84	13.31	39		3.4	AGA	7	4	6	1			
29	raji	25	G2P1L1	22	2.3	16.3	32.6	3.9	28	14.11	13.93	39		3.1	AGA		7	8	1			
30	preetha	24	primi	21	2.1	14.2	33.1	4	27	14.79	14.81	40		2.95	AGA	5	7	8	2			

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
31	kannagi	21	primi	21.4	2.4	16.1	33.4	4	26.4	14.91	15.15	39		2.85	AGA		7	8	1			
32	revathi	31	primi	20.2	2.1	14.2	33.5	4.1	27.2	14.79	15.07	38		2.7	AGA		7	8	1			
33	shobana	26	G2P1L1	21.4	2.4	15.6	33.2	4.1	26.6	15.38	15.41	35		2.25	AGA	3	6	7	1		4	
34	janaki	23	G2A1	21.1	2.1	14.3	32	3.7	26.3	14.69	14.07	39	3	3.2	AGA		7	8	1			
35	vahitha	25	G2P1L1	22.1	2.4	16.1	32.6	4	26.6	14.91	15.04	39		2.9	AGA		7	8	1			
36	hema	21	primi	22	2.3	15.6	32.5	4.1	27.8	14.74	14.75	38		2.85	AGA		7	8	1			
37	renuka	27	G3P1L1A1	21.5	2.3	15.5	33.3	4.3	28.8	14.84	14.93	39		2.85	AGA	4	5	7	2		3	
38	vimala	23	primi	20.4	2.1	14.3	32.6	4	27.4	14.69	14.6	38		2.75	AGA		7	8	1			
39	leena	26	primi	21.3	2.3	15.6	32.1	3.8	25.4	14.74	14.96	40		3	AGA		7	8	2			
40	vani	23	primi	22.1	2.5	16	32.6	4	25.1	15.63	15.94	39		2.95	AGA		7	8	1			
41	padma	23	primi	21.3	2.4	16.9	32.2	3.9	26.8	14.2	14.55	39		2.85	AGA		7	8	1			
42	malar	21	primi	21.2	2.3	17.6	33	4.1	29.8	13.06	13.76	38		2.7	AGA		7	8	1			
43	parveen	27	primi	20.4	2.1	14.1	33.2	4	27.2	14.89	14.71	39		2.95	AGA		6	7	2			
44	roselin	25	G2P1L1	21	2.1	14.3	33.4	4.1	28.3	14.69	14.49	39		2.9	AGA		7	8	1			
45	shanthi	29	G3P1L1A1	21.2	2.3	15.6	33.5	4.2	28.4	14.74	14.79	39		2.85	AGA		7	8	1			

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
46	manju	21	primi	20.5	2.1	14.9	33	4.1	28.6	14.09	14.34	38		2.75	AGA		6	8	1			
47	maria	22	primi	22	2.5	16.9	32.6	4.2	28.3	14.79	14.84	39		2.95	AGA		7	8	1		4	
48	anuradha	23	primi	21.5	2.3	15.3	33.1	4	23.5	15.03	17.02	38	2	1.95	FGR	2	4	7	1	1	3	1
49	lavanya	23	primi	21.4	2.4	16.2	32.4	3.9	26	14.81	15	39		3	AGA		6	7	1			
50	razia	26	G2P1L1	22.4	2.6	17.9	32.5	3.9	26.2	14.52	14.89	35		2.3	AGA	6	7	8	1			
51	sankari	27	G2a1	20	2.1	14.3	33	4	28.2	14.69	14.18	39		2.95	AGA		6	7	1			
52	sinthiya	21	primi	21.5	2.4	16.3	33.1	4	26.4	14.72	15.15	38	1	2.8	AGA		7	8	1			
53	jeeva	19	primi	21	2.2	15.3	32.4	3.9	26.6	14.38	14.66	39		3.1	AGA		7	8	2			
54	laila	24	G2P1L1	20.5	2.2	14.6	32.3	3.9	26	15.07	15	39		3.25	AGA		7	8	1			
55	sowmiya	24	G2P1L1	20	1.9	13.9	33	3.8	28	13.7	13.57	40		3.4	AGA		7	8	1			
56	savitha	26	primi	21.3	2.3	17.1	32.6	4.1	29.3	13.45	13.99	38		2.75	AGA		7	8	1			
57	chandra	21	primi	20	2	14	32.6	4	28.1	14.29	14.23	39		2.9	AGA		7	8	1			
58	sultana	23	primi	20.1	2	14.1	33.6	4.3	29.6	14.18	14.53	38		2.75	AGA		7	8	2			
59	shanthi	26	G2P1L1	21.1	2.3	15.7	33.5	4.2	28.6	14.65	14.69	39		2.95	AGA		7	8	1			
60	sharmila	26	G3P1L1A1	21.2	2.2	15.2	33.5	4.2	25.4	14.47	16.54	38	1	2.25	FGR	3	5	7	1	1	3	

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
61	sandhiya	20	primi	22	2.4	16.3	32.4	3.9	26.5	14.72	14.72	40		3	AGA		7	8	1			
62	vanitha	23	primi	21	2.1	14.3	33.1	4	27.2	14.69	14.71	38		2.9	AGA		7	8	1			
63	kavitha	23	primi	22.2	2.5	15.9	33.2	4	26.2	15.72	15.27	38		2.9	AGA		7	8	2			
64	suguna	28	G2P1L1	20.5	2.1	14	32	3.9	25.9	15	15.06	36		2.5	AGA		5	7	2			
65	gomathi	21	primi	21.4	2.3	16	32.5	4.1	28.8	14.38	14.24	39		3	AGA		7	8	1			
66	sundari	21	primi	21.3	2.3	16.1	32.3	4.1	29.5	14.29	13.9	39		2.9	AGA		7	8	2			
67	devi	24	primi	22.3	2.5	19	32.5	4	29.4	13.16	13.6	38		3.3	AGA		7	8	2			
68	sheela	27	G2P1L1	20	2.1	14.3	33	4.1	27.8	14.69	14.75	39		3	AGA		7	8	1			
69	malathi	21	primi	21.1	2.3	16.5	33.6	4.3	30.1	13.94	14.29	37		2.7	AGA		7	8	1			
70	surya	24	primi	21.2	2.2	14.2	34	4.3	28	15.49	15.36	40		3.1	AGA		7	8	2			
71	vaishnavi	23	primi	21	2.3	16	32.6	4	27.6	14.38	14.49	38		2.9	AGA		4	8	1			
72	pavithra	25	G2P1L1	20.6	2.1	14.1	32.2	3.9	26.4	14.89	14.77	38		3	AGA		7	8	1			
73	girija	23	primi	20	20	16.2	33.3	4.1	30.6	12.35	13.4	39		3	AGA		7	8	1			
74	geetha	23	primi	22.1	2.3	14.7	32.6	4	26.1	15.65	15.33	38		2.75	AGA		7	8	1			
75	dhanam	23	primi	21.5	2.3	16.1	33	4	23.4	14.29	17.09	39	1	1.8	FGR	10	4	6	2	1	2	

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
76	mohana	21	primi	21.4	2.1	14.1	34	4.2	28.1	14.89	14.95	39		2.9	AGA		7	8	1			
77	prabha	25	G2P1L1	22	2.3	15	33.4	4.2	27.6	15.33	15.22	39		2.95	AGA		7	8	1			
78	roksana	20	primi	21.3	2.4	16.3	32	3.9	23.8	14.72	16.39	38	2	2.2	FGR		7	8	2	1	4	
79	sivagami	29	G2P1L1	20.1	1.9	13.8	32.5	4	29.2	13.77	13.7	39		3.7	AGA		6	8	1			
80	indra	30	G3P2L2	20.1	1.9	14.7	33	3.8	29.3	12.92	13.29	39		3.1	AGA		7	8	1			
81	kumutha	23	primi	20.4	2.1	14.2	32.6	3.9	27.2	14.79	14.34	37		3	AGA		7	8	1			
82	soniya	25	G2P1L1	20	2	14.1	34	4.2	30	14.18	14	39		3.4	AGA	2	7	8	1		3	
83	gowri	23	primi	20.2	2	14	34.1	4.3	29.7	14.29	14.49	39		3.2	AGA		7	8	1			
84	mala	21	primi	20.1	1.9	13.6	32.5	3.9	28.7	13.97	13.59	40		3.5	AGA		7	8	2			
85	rachel	22	G2P1L1	22	2.3	15.9	33.4	4.1	23.7	14.5	17.29	37	1	1.75	FGR	4	6	7	2	1	4	
86	sridevi	30	G3P2L2	21.5	2.4	16.3	33.1	4	27	14.72	14.81	38		3.2	AGA		7	8	1			
87	megala	21	primi	22	2.5	18.8	32.6	4	26.6	15.29	15.04	38		3.1	AGA		7	8	2			
88	usha	26	G2P1L1	20	2	14	33.5	4.1	28.5	14.29	14.39	39		3.4	AGA		7	8	1			
89	dhanam	32	primi	21.6	2.5	16	34	4.3	27.8	15.63	15.47	39	1	3.2	AGA		7	8	1			
90	pushpa	24	primi	20	1.9	13.8	33.6	4.2	30.5	13.77	13.77	39		2.9	AGA		7	8	1			

S.No	name	age	Obstetric Code	GA1	TCD1	AC1	GA2	TCD2	AC2	TCD1/AC1 Ratio (%)	TCD2/AC2 Ratio (%)	GA at Delivery	Risk Factors	BW in kg	In utero growth status	NICU Admission	APGAR 1	APGAR 5	Mode of Delivery	Type of FGR	Perinatal Morbidity	Perinatal Mortality
91	vasanthi	26	G3P1L1A1	21.3	2.4	16.2	32.3	3.9	26.9	14.81	14.5	40		3	AGA		7	8	1			
92	rani	24	primi	21.4	2.3	15.1	32.4	3.9	26	15.23	15	39		2.9	AGA		7	8	2			
93	bhuvana	20	primi	20.1	2.1	14	33.3	4.1	27.9	15	14.7	39		2.95	AGA		7	8	1			
94	mangai	29	primi	20.4	2.2	13.4	32.3	3.8	23.9	16.42	15.9	36	2	1.8	FGR	3	5	7	2	2	5	
95	rajathi	20	primi	21	2.3	15.5	34.3	4.3	29.2	14.84	14.73	39		2.95	AGA		7	8	1			
96	eswari	24	primi	20.3	2.1	14.5	32	3.9	26.7	14.48	14.61	39		3	AGA		7	8	1			
97	ramya	23	G2P1L1	22	2.5	16.5	32.4	4	26.6	15.15	15.04	40		3.1	AGA		7	8	2			
98	anitha	27	G2P1L1	21	2.1	14	32.5	4	26.8	15	14.93	39	1	3.2	AGA		7	8	2			
99	mohana	22	primi	20	2	14	34.2	4.2	29.4	14.29	14.29	39		3	AGA		7	8	1			
100	rihana	23	primi	21.5	2.3	15.9	34.2	4.3	30.4	14.5	14.14	39		2.8	AGA		7	8	1			

Key to Master Chart

KEY TO MASTER CHART

1	**Obstetric Code**		
		G	**Gravida**
		P	**Para**
		L	**Live**
		A	**Abortion**
2	**GA**		**Gestational Age**
		1	**20-22 weeks**
		2	**32-34 weeks**
3	**TCD**		**Transverse Cerebellar Diameter (in cm)**
		1	**20-22 weeks**
		2	**32-34 weeks**
4	**AC**		**Abdominal Circumference (in cm)**
		1	**20-22 weeks**
		2	**32-34 weeks**
5	**TCD1/AC1 Ratio (%)**		**TCD/AC Ratio (%) at 20-22 weeks**
	TCD2/AC2 Ratio (%)		**TCD/AC Ratio (%) at 32-34 weeks**
6	**Risk Factors**		
		1	**Preeclampsia**
		2	**Oligohydramnios**
		3	**GDM**
		4	**Chronic Hypertension**
7	**BW**		**Birthweight**
8	**In utero**		

	growth Status		
		AGA	**Appropriate for Gestational Age**
		FGR	**Fetal Growth Restriction**
9	**NICU Admission**		**Neonatal Intensive Care Unit Admission**
10	**APGAR**		
		1	**1 minute**
		5	**5 minutes**
11	**Mode of Delivery**		
		1	**Vaginal**
		2	**LSCS**
12	**Type of FGR**		
		1	**Asymmetrical**
		2	**Symmetrical**
13	**Perinatal Morbidity**		
		1	**Asphyxia**
		2	**Meconium Aspiration Syndrome**
		3	**Hypoglycemia**
		4	**Hypocalcemia**
		5	**Hypothermia**